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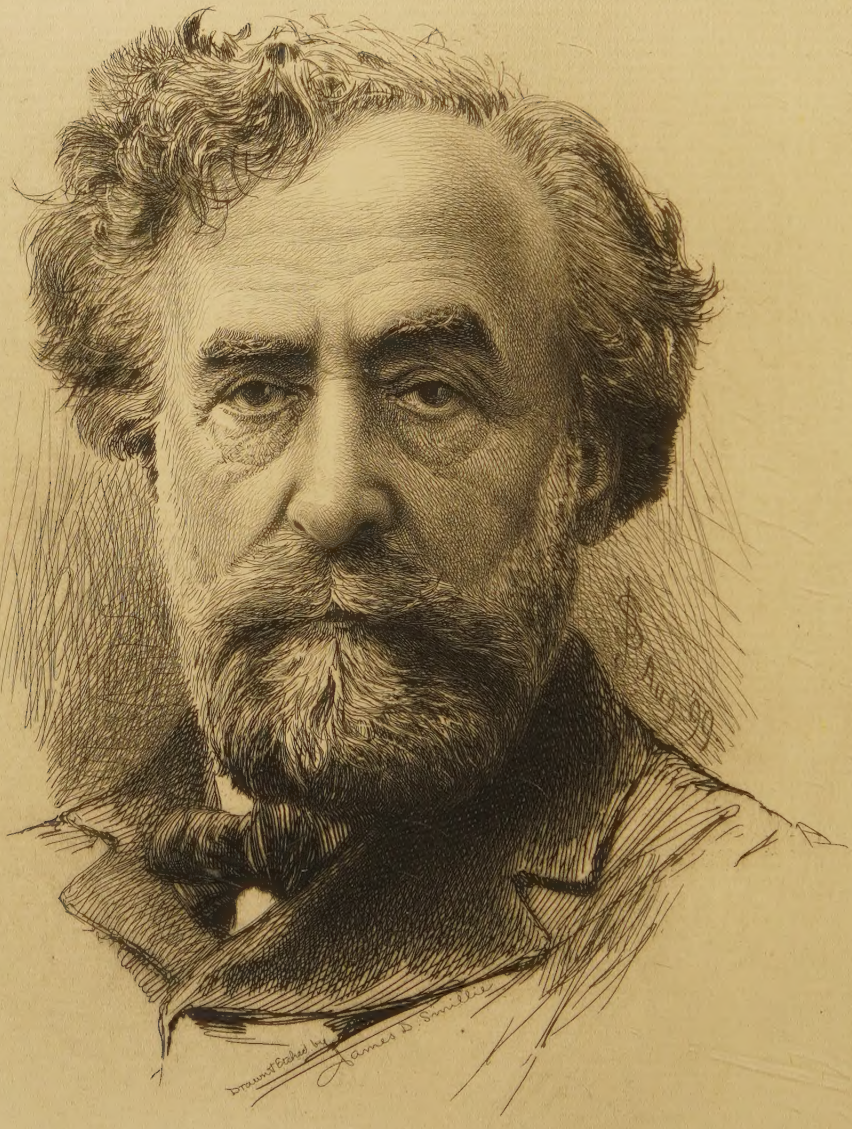

a Jacobi

INTERNATIONAL CONTRIBUTIONS

TO

MEDICAL LITERATURE

"FESTSCHRIFT"

IN HONOR OF

ABRAHAM JACOBI, M.D., LL.D.

TO COMMEMORATE THE

SEVENTIETH ANNIVERSARY

OF HIS BIRTH

MAY SIXTH, 1900

The Knickerbocker Press

NEW YORK

1900

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PREFACE.

IN reading Mark Twain's description of life in Berlin, the American reader is particularly impressed by the account of the reception tendered to Virchow on his seventieth birthday. Honors of which the highest in the land might have been proud were heaped upon that illustrious scientist and statesman. Few physicians can hope to attain the position of Virchow or receive such homage. It is a fact, however, that men eminent in special branches of medicine, great teachers and clinicians, are honored by their former pupils, their colleagues, and their university in a similar manner, but with less pomp and éclat. In addition to the festivities and the professions of good-will and esteem, a more enduring honor is often awarded. Pupils and professional friends show their appreciation by dedicating in his honor a volume of essays pertaining to his particular branch of medicine. Such a book has been termed a "Festschrift." No word in the English language conveys the same idea. The terms Jubilee Volume, Memorial Volume, Medical Symposium, Dedicatory Volume, were considered, but all seemed awkward and inexpressive of the object of the work. It seemed wise to the committee, therefore, to employ the German title, which is brief and expressive. The "Festschrift" is usually presented at the completion of the twenty-fifth or fiftieth year of professorship or upon the anniversary of the seventieth birthday.

Our great teachers in America, though their professional standing be of the highest, and their character without reproach, receive but scanty recognition on such occasions. As they pass from the stage of active life and are followed by younger men, their memory becomes too often but a tradition or is entirely lost. This is deeply to be regretted. The German custom of perpetuating the memory of its great men by some marked expression of appreciation and esteem on the part of professional brethren is one eminently worthy of adoption in America. The brilliant attainments and strong personality of Dr. Jacobi induced a number of professional friends and former pupils to feel that they were more than warranted in instituting such a testimonial in his honor. Indeed, the fact that he would be the first

to receive such an honor seemed especially appropriate. In March, 1899, therefore, the movement which has terminated in the production of this volume was set in motion. A working committee was appointed and active measures were at once taken. Many authorities, both here and abroad, heartily endorsed the project. Several, unable to prepare essays, sent their congratulations and expressions of esteem, among whom are the following:

Clifford Allbutt says: "Dr. Jacobi is an old and dear friend of mine, so that I the more rejoice in the distinction which his personal and scientific qualities have won for him among all English-speaking people."

Henry Ashby expresses his great appreciation of Dr. Jacobi's work and the pleasure it has always given him to meet him.

William B. Cheadle sends his most hearty congratulations and good wishes.

Reginald H. Fitz says that Dr. Jacobi well deserves all the honors the profession can bestow.

Marfan: "Je m'associe de tout cœur à l'hommage que vous voulez rendre à notre vénéré collègue, le Prof. Jacobi."

J. H. Musser expresses his heartiest approval of the project.

Nothnagel writes: "Da ich Jacobi sehr hoch schaetze, würde es mir eine grosse Freude sein, mich an derselben zu betheiligen."

A. J. C. Skene says: "I have all my professional life been a profound admirer of that great and good man."

W. S. Thayer approves most heartily of any honor which the profession may see fit to confer upon Dr. Jacobi.

William H. Welch says: "I consider Dr. Jacobi one of the greatest ornaments of the medical profession in this country. He has done inestimable services not only in New York, but for the profession of the whole country."

Dr. Jacobi's career has been one of ceaseless activity. At eighteen years a participant in the struggle for free Germany, in '48, he was imprisoned again and again, and for two years suffered the greatest hardships. He was a co-worker and has been a lifelong friend of such men as Schurz, Siegel, Kunlich, Ottendorfer, Binder, Von Holst, and Krackowizer.

A graduate in medicine in Bonn in 1851, he came to New York in 1853. In 1857, he became a Lecturer on Infantile Pathology in the College of Physicians and Surgeons. In 1860, the first chair upon Diseases of Children established in America was instituted in the New York Medical College with Dr. Jacobi as professor. In 1865, he became Professor of Diseases of Children in the University of New York, but

resigned in 1870 to become Clinical Professor of the Diseases of Children in the College of Physicians and Surgeons, and thirty years later he is still found in this school expounding the most recent teachings in scientific medicine.

In 1895, his name was mentioned in connection with a clinical professorship in one of the largest schools of Germany. This honor he was constrained to decline, feeling that the ties which bound him to America were too strong to be broken. Three years later he was honored by one of America's greatest universities, when the degree of Doctor of Laws was conferred by the University of Michigan, in recognition of his broad scholarship and scientific and literary attainments.

The first to inaugurate systematic and special clinics in diseases of children in New York, he has been continuously active in clinical work for twoscore years. To-day, he has the satisfaction of seeing the fulfillment of his fondest hopes in the universal recognition of pediatrics as a distinct branch of medicine. In 1898, the first ward in this country designed primarily for the clinical teaching of pediatrics was opened in Roosevelt Hospital and was named the "Jacobi Ward for Clinical Instruction." "The opening of this ward," says a prominent medical journal, "designed as it is primarily for bedside instruction of medical students, marks an epoch in the teaching of diseases of children. It seems very appropriate that the first ward designed for this purpose should bear the name of the man who has inaugurated so many advances in the teaching and practice of pediatrics. It is a most fitting tribute to his life of devotion to that department of medicine."

In 1857, Dr. Jacobi was one of the founders of the German Dispensary and in 1868 of the German Hospital. For many years he was consulting physician to the Board of Health, and to the Hood Wright Memorial Hospital, and a visiting physician to the Nursery and Child's Hospital. He has been a physician to Mt. Sinai Hospital since 1860, to Bellevue Hospital since 1873, and to Roosevelt Hospital since 1898. He is also consulting physician to the Babies' Hospital, the Skin and Cancer Hospital, the Orthopedic Hospital, and the Hackensack Hospital.

As a society worker, Dr. Jacobi has ever been active and prominent. He was president of the New York Obstetrical Society and the Pathological Society in 1864, of the County Society in 1870 and 1871, of the State Medical Society in 1882, of the New York Academy of Medicine from 1885 to 1889, of the American Pediatric Society in 1889, of the Association of American Physicians and the German Medical Society

of New York in 1896, and the American Climatological Association in 1900. He was a prime mover in the founding of the Section on Pediatrics of the American Medical Association in 1880, of the Pediatric Section of the New York Academy of Medicine in 1885, and of the American Pediatric Society in 1888. He is an honorary member of medical societies in Würzburg, Berlin, Buda-Pest, Paris, Philadelphia, Boston, and Louisville.

For more than forty years, the German-speaking physicians of New York and vicinity have held their monthly meetings under his roof. The attendance is large and the scientific work of high order. A broad spirit of good fellowship is fostered by the discussions between the older and younger men, the specialist and the family practitioner.

In the international meetings, no one else has done so much to promote the interest and widen the fame of American medicine. He held a prominent position in the International Congress at Copenhagen, Berlin, and Rome, and he has been nominated an honorary president of the Congress to be held in Paris in 1900.

In 1858, Dr. Jacobi published his first paper on pediatrics, the same year in which J. Lewis Smith published his first paper on children. Since that time his contributions to medical literature have been legion. One of the most notable among these was a paper on diphtheria in 1860. This was the first paper published on the Atlantic Coast upon this subject. It was followed by many papers on the same subject until 1880, when his *Treatise on Diphtheria* appeared. The contributions on "Midwifery and the Diseases of Women and Children" by A. Noeggerath and A. Jacobi, 1859, received much attention. "Dentition and its Derangements" appeared in 1862; the "Intestinal Diseases of Infancy and Childhood," in 1887; "Hygiene und Pflege der Kinder," in *Gerhardt's Handbuch*, vol. i., 1876, deserve to be remembered. His last important publication was his *Therapeutics of Infancy and Childhood*, published in 1895, with a second edition in 1897. In 1893 there were published two handsome volumes of miscellaneous essays and addresses, covering a great variety of subjects, many of them not strictly medical. It is much to be regretted that they were not designed for large circulation.

Dr. Jacobi's fame does not rest alone upon his greatness as a teacher and writer. He has taken an active interest in all that is best in medicine and has, withal, been an active and public-spirited citizen. As author, practitioner, hospital physician, and society worker, he has ever been in the harness. In all these various departments of professional endeavor, he has been a leader and has never been content to be a servile follower.

As president of the New York Academy of Medicine at a most critical period in its history, Dr. Jacobi was to an unusual degree instrumental in promoting not alone the present, but the future interests of the medical profession of the metropolis. The new building for the Academy (now free from all indebtedness) was brought about mainly through his efforts. His work in behalf of the library alone can hardly be estimated. It is largely due to him that among medical libraries in America it is now second only to that of the Surgeon-General at Washington. As president he was largely instrumental in establishing a number of special sections. Not only did he preside at the general meeting, but the sections were frequently graced by his attendance and edified by a discussion of the papers presented. Many of the younger men owe their first introduction and a part of their subsequent success to his paternal guidance. Always anxious to induce the rising practitioners to take an active part in the work of the Academy, the success of the infusion of the younger element is evident in the larger attendance and the improved character of work.

As first professor of the diseases of children in America, as tireless writer and teacher, as first president and active worker of the American Pediatric Society, he has unquestionably done more than any other man to place pediatrics upon a firm and enduring basis. It is upon this portion of his work that his fame will chiefly rest. It must be remembered that he never regarded pediatrics as a new specialty, but rather as cognate to general practice and an intimate associate of all departments of medicine. "The Relations of Pediatrics to General Medicine," the president's address delivered before the American Pediatric Society in 1889, and the "Introductory" to *Keating's Cyclopædia* were admirable presentations of the true status of pediatrics.

During his whole career Dr. Jacobi's influence has been exerted towards improving the tone and morale of the medical profession, and advancing its code of practical ethics. He aided in abolishing the written code in New York, believing it could do nothing to improve the conduct of gentlemen. He has stood firm and unmoved against the rising tide of commercialism. Thoroughly approving of specialism, he has strenuously opposed the exuberant growth of immature specialists, and has steadfastly upheld the rights of the general practitioner. He has never ceased to attend medical meetings, local, state, national, and international, and to participate in all that is of vital interest to medical men.

But it is not alone as a medical man that Dr. Jacobi is worthy of honor. He has also sought to promote the welfare of his fellow-men, as a man and a citizen. He has taught the propriety of physicians

taking an interest in public affairs, and has exemplified his teaching by taking an active part in many matters of civic and political importance, serving on public committees, addressing legislative bodies, and urging questions of public policy. A patriot in his youth and ever retaining his love for the Fatherland, he has been a true and faithful son of the land of his adoption, appreciative of her ideals, loyal to her traditions, and faithful to his duties as a citizen. So true, indeed, has he been to each, that Fatherland and fosterland, as well as other nationalities, unite in doing him honor by offering him the tribute of this volume, and expressing the hope that, though threescore years and ten have passed since his natal day, ripe in years and experience, still young in heart and ardent in work as of yore, he may be spared for many years to reap in profusion the fruits of an arduous and well-spent life.

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APPRECIATION OF DR. JACOBI'S WORK AT ALBANY.

By A. VANDER VEER.

FOR many years there has been a tradition, among the older physicians in Albany, or impression, that when Dr. Jacobi first came to this, his adopted country, he had some thought of locating in Albany. Whether this belief was correct or not, certain it is that with the physicians here, and citizens in every walk in life, he has always held a very close and dear relationship. Few men have had so many loyal and true supporters, outside of his own city, as has Dr. Jacobi in the capital city of this State. In the profession, whenever it was deemed advisable to approach the Legislature for the improvement of our laws, to bring our calling up to its present high plane of usefulness and standing, one of the first men in mind, and the first to be consulted, has been Dr. Jacobi. He has been ever faithful in his response. No effort has been too great for him to make. If coming from New York City was necessary, to appear before some committee in the Legislature, he has ever been ready to respond, and the formulation of some of the present laws, which are of so much value to us in maintaining the dignity and self-respect of our profession, are due to his personal efforts. When in the process of the evolution of our medical laws it was thought wise for the University of the State to grant the degree of M.D. to candidates on examination, the writer is thoroughly conversant with the facts whereby the Regents at once looked to Dr. Jacobi as the leader, and of this board he was by acclamation appointed president. No State officer has ever discharged his duties more conscientiously than has Dr. Jacobi, while presiding over this State Board of Medical Examiners.

The writer's recollection is that he never neglected a meeting, was always in attendance, gave his close attention to the preparation of questions, and methods of conducting the examinations, and the system of markings. By no possibility could any candidate pass who did not come up to the regular requirements. The successful working of

this board, being so just and fair in all its deliberations, was a mighty lever in the passing of the laws that organized the three State boards, which has helped to place this State in such a proud position before this country ; also, as shown by the recent publication of the Regents' office, to command the respect of foreign countries.

In the shaping of the policy, and successful career of the State Medical Society, Dr. Jacobi has been one of the most prominent and reliable factors. His attendance upon the annual gatherings here is always looked forward to with pleasure on the part of his many admirers. His papers are always listened to with the utmost attention, and no one leaves the room when Dr. Jacobi is discussing a paper on any subject of interest to the medical profession. In the latter he is particularly strong. He always gives the evidence of preparatory discipline of years, and which so often gives him the advantage when discussing intricate and important points in pathology, and especially in the diseases of children.

At our different banquets he is always the centre around which gathers the closest attention, the most emphatic applause. His remarks are always appropriate. Some good lesson can be drawn from his utterances at all times. Dr. Jacobi treats the members of his profession as do many statesmen the public. He takes them into his confidence, he gives them his reasons for arriving at certain conclusions, he instructs them in such a manner that they can see clearly and intelligently what he is aiming at. When not the original investigator himself he presents his observations by reference to such writers as are authorities, giving evidence of his being in good company and familiar with all the best teachings of the day. Dr. Jacobi has always exhibited that great virtue modesty. His has been no cheap and transparent reputation. The echoes that have come to him from his professional life have been strong and wholesome, as an endorsement from his fellow associates, and from the public at large.

Dr. Jacobi exhibits rare combinations in many ways. Whenever the State Medical Society required subscriptions for any extra expense he has been the first to head the list, and with a liberality that has always commanded our respect and admiration. If any embarrassment arises, in which it becomes necessary to raise a certain sum, for some particular purpose, he always responds willingly and cheerfully, without any criticism, but throws himself fully into the arms of those with whom he has always been associated, trusting their methods and their earnestness with the most implicit confidence.

His great range of professional education, his time, his money have been given freely to the profession of this State and country. When

we turn aside the veil that shuts out, to a certain extent, the general observations and workings of our national associations, our special societies, our medical congresses, we see a striking illustration of the national esteem in which Dr. Jacobi is held. His friends have found great pleasure in the inquiries that are always made on the first day of the gatherings of these various bodies as to whether Dr. Jacobi would be there. "Will he present a paper, will he discuss this or that subject, will he serve on this or that committee, and give the necessary time required for the sittings of such a committee?" It has afforded the writer much happiness, on many occasions, to reply "yes," and to emphasize that "if Dr. Jacobi be appointed, and consents to serve on any committee, you may be sure he will give of his time, his pen, and his brain work, all that may be required, and, if necessary, of his money."

In the history of our State Medical Society the writer has been the observer of many a preparation for the annual meetings, and, when the outlook was dark, and hard work ahead, no one responded more promptly than did Dr. Jacobi. Few there are whose names have been so often quoted when there was a hard problem to solve. In the preparation of many programmes it has always been a joy to observe the selection of certain names, names of those who could be relied upon to present papers thoroughly abreast of the times, who were willing to do committee work, to make reports, and no one name has ever been more prominent in this connection than that of Dr. Jacobi; therefore, we may say that in medical legislation, in committee work of all kinds, in fearless interpretation of the code, in quiet consultation, when great questions were being considered, and the welfare of the State Medical Society held in the balance, Dr. Jacobi's judgment and conclusions always received the most profound endorsement.

When some public or educational institution was to be dedicated no one name was considered more often, and looked to with more earnestness, regarding invitation and acceptance of the same, than was, and is, Dr. Jacobi's. The Trustees of the Bender Hygienic Laboratory, the Faculty of the Albany Medical College, and citizens of Albany will ever hold him in high regard. He gave us his time and his talents when dedicating that great gift, the Bender Hygienic Laboratory, the outgrowth of the liberality and public spirit of Mr. Matthew W. Bender, and when the day arrived for the dedication of this gift, Dr. Jacobi responded with no hesitancy or apology, but gave such a fine address as brought forth the enthusiasm of all who are connected with this advanced pathological institution. Then did Dr. Jacobi show his great range of student life, of careful, continuous reading, of close observation, and his thorough knowledge of pathological

questions. His address was admirable and impressive, and was so full of wise suggestions, and close observations upon the pathology of the day, that it has been a guidance and aid in the building up of this laboratory. No one could have made an address more appropriate, for the student and graduate profession.

This but faintly suggests the results of thirty years' acquaintance and observations of Dr. Jacobi's work.

CHOREA.

By M. ALLEN STARR, M.D., LL.D.

FROM the opening of the Vanderbilt Clinic in May, 1888, until the 15th of November, 1899, fourteen hundred cases of chorea had been treated in the Nervous Department. The history obtained of each case has been sufficiently accurate and the physical examination of each patient sufficiently reliable to warrant a few statements in regard to the etiology of the disease. Uniformity in the record of the cases has been secured by their entry in a book especially prepared for their history. For these reasons the statistics which are here presented appear to have a certain value.

Of the 1400 cases, 460 were males and 940 were females. The age of onset of the disease was as follows:

3 yrs..... 8	9 yrs..... 155	15 yrs..... 65
4 yrs..... 19	10 yrs..... 145	16 yrs..... 52
5 yrs..... 46	11 yrs..... 123	17 yrs..... 35
6 yrs..... 83	12 yrs..... 114	18 yrs..... 26
7 yrs 104	13 yrs..... 128	19 yrs..... 13
8 yrs..... 150	14 yrs..... 98	20 yrs..... 14

Between 21 and 30 yrs..... 29.

The class of children affected is well known to be the poor, badly fed, badly nourished, badly housed class, and this fact becomes noticeable when the relatively small number of cases of children suffering from chorea seen in private practice is compared with the number appearing at the Clinic. It is quite the exception to see the disease in healthy, well fed children, and the cases observed in private practice have been in the majority of instances traced to hereditary predisposition to nervous conditions rather than to the surroundings of the child.

In 835 of the Clinic cases the record shows a condition of anæmia present. This statement is made on the basis of the pale, thin appearance of the child, pallor of the mucous membranes as well as of the skin being frequently noted, but it is not based on any blood count or estimate of per cent. of hemoglobin. Burr (*University Medical*

Magazine, Dec., 1896), while noting the appearance of anæmia in a number of cases, found the blood normal, corpuscles not wanting, and hemoglobin from 60 % to 85 %, hence it is possible that the anæmic appearance of these children may not be actually attended by true anæmia in all cases. Their appearance, however, is noticeably anæmic. There is no evidence of heredity to be gathered from the histories, it being a very rare exception to learn that either parent had suffered from the disease, and as a rule the parents were not found to be nervous or to be afflicted with nervous diseases, such as epilepsy or insanity. In 53 cases, brothers or sisters had chorea at the same time as the patient.

The personal history of the children showed that there was no definite relation between the occurrence of the infectious diseases of childhood and the development of chorea. For though a majority of children had had such diseases, they did not precede the chorea immediately, or appear to stand in any causal relation to it. It may be of interest to note the liability of children to the various infectious diseases. This is shown in a series of 1000 cases. Of these 575 had had measles, 245 whooping-cough, 210 scarlet-fever, 115 diphtheria, 27 malaria, 6 typhoid fever, and 4 meningitis.

It is extremely difficult to ascertain any definite cause for chorea. In 285 cases a history of fright or sudden alarm of a sufficiently intense character to produce a mental shock was obtained. In a few cases overstrain at school was supposed to be a cause, but the number was insignificant. The general malnutrition appeared to be the most constant element in the etiology.

The relation of chorea to rheumatism has been studied by many authorities, and the statement has often been made that chorea, rheumatism, and endocarditis are varying results of the same toxic agent in different cases. Of the 1400 cases here collected, 290 presented a distinct history of true rheumatism, with swollen, painful, red joints and disability for some days. Many more had complained of "growing pains," but as it is doubtful whether these pains, not necessarily located in the joints, were of rheumatic origin, they are not included in the list of rheumatic cases. One or both parents were found to have had rheumatism in 263 cases.

In a collection of cases of chorea made by me from fifteen different authorities in different countries, numbering 2000 cases (see L. Starr's *Text-book of Diseases of Children*, Art. "Chorea"), I found that 26 % presented a history of rheumatism. It will be noted that of the 1400 cases here recorded, but 21 % presented a rheumatic history. This is a smaller percentage than that obtained by many authorities.

An examination of the heart has been carefully made in almost every case. In 175 cases an organic heart murmur was found, which persisted after the cure of the chorea. In 123 cases a functional heart murmur was found which disappeared after the cure of the chorea. In 871 cases a statement of physical examination with the record of "no heart murmur" was found. In the other cases no record was made.

It is quite interesting to note the month of onset in the disease, inasmuch as attempts have been made to establish some relation between the occurrence of chorea and the season of the year.

The attack developed in	January	in 85 cases.
	February	98
	March	111
	April	131
	May	128
	June	112
	July	118
	August	107
	September	77
	October	54
	November	47
	December	61

In my opinion the reason for the appearance of the disease chiefly in the spring of the year, March, April, and May, is that at this time the confinement of the children in the house in bad air, and the inability to obtain outdoor exercise during the winter results in the culmination of the malnutrition, which is the chief predisposing cause of the disease; while the lesser number in the summer and fall indicates a better nutrition of children who have passed most of the time out-of-doors.

It was noted that in 951 cases the choreic movements were general in their distribution, and in 449 they were unilateral, a right unilateral chorea being slightly more frequent than a left. In addition to the choreic movements, mental irritability was distinctly noted in 827 cases; speech was affected in 556; sleep was bad in 415.

There is a well marked tendency to relapses in the history of chorea. This is not surprising, inasmuch as it is evident, from our view of the causation, that the children are subjected to the same deleterious influences every winter; hence recurrences of the disease might be expected. The frequency of such recurrences is shown from the fact that out of the 1400 cases, one quarter had had previous attacks.

231 had had one previous attack.
56 had had two previous attacks.
40 had had three previous attacks.

18 had had four previous attacks.
6 had had five previous attacks.
9 had had six previous attacks.
1 had had seven previous attacks.
3 had had eight previous attacks.
1 had had nine previous attacks.

It was noticed that in almost all of these cases the various attacks occurred at the same time of the year and under practically identical circumstances. In private practice I have not observed such a tendency to recurrences, possibly because better care and nutrition and a change of climate have been insisted upon, and all causes of nervous tension have been carefully removed for at least two years after the occurrence of the original attack. Such care being impossible among the poorer classes, recurrences are less easily warded off.

Of the remedies prescribed for chorea, arsenic appears to be the one of greatest value, and it is my rule to increase the doses of the drug rapidly up to the point of physiological effect and then to reduce them slowly, keeping the child just upon the verge of intoxication by the arsenic. The susceptibility of children to arsenic is very different in different individuals. In two cases severe general arsenical neuritis appeared during the course of treatment, although these two children were not at the time taking as much of the drug as was administered without ill effects to many others. Both cases recovered. Antipyrine appears to be the remedy next in value to arsenic, but exalgin, phenacetin, bromide, chloral, and paraldehyde have been tried with very little effect.

It appears to me that less dependence is to be placed upon any drug treatment of the disease than upon an immediate change of air and surroundings. I have seen many very bad cases recover almost entirely within two weeks after being sent to the seashore or to one of the country homes of the children's hospitals of this city, when they had resisted treatment for months by all forms of drugs.

ÜBER "GENERALISIRTE VACCINE."

VON PROFESSOR E. HENOCH (DRESDEN).

UNTER dem Namen "generalisirte Vaccine" versteht man bekanntlich den nach der Impfung am Arm erfolgenden Ausbruch von Vaccinepusteln an verschiedenen Theilen der Körperoberfläche, nicht blos an den geimpften Stellen. Die Literatur ist nicht gerade reich an Fällen dieser Art, aber man kann wohl sagen, dass fast alljährlich die Zahl derselben sich vermehrt. Ich will nun gleich bekennen, dass mir selbst kein sicheres Beispiel dieses ungewöhnlichen Impfereignisses vorgekommen ist, gebe aber gern zu, dass meine eigenen Erfahrungen über die Impfung und ihre Folgen keineswegs ausreichend sind, um ein Urtheil über das Vorkommen dieser Anomalie in negativem Sinne zu begründen. Denn von vornherein lässt sich die *Möglichkeit* einer solchen Verallgemeinerung der Vaccineeruption gewiss nicht in Abrede stellen. Unter anderen bietet ja doch die Syphilis das nächstliegende und ausgezeichnetste Beispiel dieser Art. Die Sache liegt allerdings so, dass was bei der Syphilis Regel ist, bei der Vaccine nur als Ausnahme vorkommen würde, eine Thatsache, die man nur durch eine besondere Eigenschaft des Virus der letzteren, etwa eine geringere Virulenz, erklären könnte.

Dass ein, wenn auch nur geringer Theil der in der Literatur mitgetheilten Fälle von "generalisirter Vaccine" gut beobachtet und richtig beurtheilt sein mag, will ich daher durchaus nicht bestreiten. Der grössere Theil aber ist meiner Überzeugung nach nicht richtig beurtheilt worden, und dürfte vor einer strengen Kritik nicht Stand halten. Es sei mir gestattet, zunächst einen Fall mitzutheilen, der sehr geeignet war, bei mir die ersten Zweifel in dieser Angelegenheit zu erwecken.

Vor einer Reihe von Jahren wurde ich in Berlin zu einem 8 Monate alten Kinde gerufen, welches nach der Diagnose des Hausarztes und eines bereits consultirten Dermatologen seit etwa 3 Tagen an den *Pocken* erkrankt sein sollte. Das Kind war noch nicht vaccinirt worden und abgesehen von einem in Form der sogenannten Crusta lactea bestehenden *Eczema impetiginosum* des Gesichts, immer gesund gewesen.

Ich fand das ganze Gesicht bis zur Haargrenze herauf stark geschwollen, beide Wangen und Stirn mit zum Theil blutig infiltrirten Eczemborken bedeckt, die ganze Umgebung derselben in einer etwa 3 Centimeter breiten Zone mit dicht aneinander stehenden linsengrossen, meistens mit einer centralen Delle versehenen Pusteln besetzt und darüber hinaus noch stark geröthet, die Augenlider beiderseits roth und so geschwollen, dass ein Einblick in die übrigens normale Conjunctiva nur mühsam zu gewinnen war, die Occipital- und Cervicaldrüsen hie und da mässig angeschwollen. Abgesehen von einem starken Juckreiz schien das Kind gesund zu sein, insbesondere war kein Fieber vorhanden, der Appetit (Ernährung mit der Flasche) ungestört.

Dass ich beim ersten Anblick des Kindes ebenfalls an Variola dachte, ist begreiflich, aber schon der Mangel des Fiebers und die Euphorie des Kindes machte mir die Diagnose der Collegen bedenklich, noch weit mehr aber die Beschaffenheit der Haut des Rumpfes und der Extremitäten. Denn mit Ausnahme von etwa 4 bis 5 bereits verschorfter Pusteln in der Umgebung der Handgelenke, erschien die Haut durchweg frei von jedem Exanthem. Dazu kam, dass jede Uebertragung von Variola- oder Vaccinevirus mit Sicherheit ausgeschlossen werden konnte. Bei dieser Sachlage musste ich mich mit Entschiedenheit gegen die Annahme von Variola aussprechen, stellte vielmehr die Diagnose: *Eczema acutum*, hervorgerufen durch mechanische Reizung eines chronischen Eczema in Folge von Kratzen, dessen blutige Spuren im Gesicht noch hie und da erkennbar waren. Der weitere Verlauf gab dieser Ansicht Recht, denn einfache laue Fomentationen des Gesichts mit Bleiwasser genügten, das scheinbar variolöse Krankheitsbild binnen wenigen Tagen in das *vor* der acuten Steigerung bestandene der Crusta lactea zurückzubilden.

Dieser Fall, der *anscheinend* mit meinem eigentlichen Thema nichts zu thun hat, lehrt also, dass ein bis dahin in gewöhnlicher Weise bestehendes chronisches Eczem unter gewissen Umständen plötzlich eine acute Form und eine Beschaffenheit annehmen kann, die einer Pockenerkrankung so ähnlich ist, dass selbst dermatologisch erfahrene Spezialisten dadurch getäuscht werden können. Seit dieser Zeit habe ich noch mehrfach, auch in der Klinik, ganz analoge Fälle beobachtet, und fast immer die Ursache in der mechanischen Insultirung des Eczems durch Kratzen gefunden, wobei nicht ausgeschlossen ist, dass dabei auch infectiöse Keime (Eitercoccen) durch die Nägel des Kindes auf die wunde Fläche übertragen werden.

Die äusserliche Aehnlichkeit der das Eczem umgebenden Pustelzone mit dicht stehenden gedellten Variolapusteln war in der That bei

diesem Kinde so auffallend, dass eine Täuschung im ersten Augenblick begreiflich erschien. Aber eine noch so grosse äussere (anatomische) Aehnlichkeit zweier Krankheitsprocesse kann doch niemals ihre Identität begründen, wenn nicht andere antiologische oder klinische Motive diese bestätigen. Die Nichtbeachtung dieser Thatsache hat wiederholt zu ganz falschen Auffassungen Anlass gegeben, z. B. zu der Confundirung der bekannten Rachennekrose beim Scharlach mit der ächten Diphtherie. Pusteln mit deutlichen Dellen kommen nicht der Variola und der Vaccine allein zu; sie treten auch gar nicht selten hie und da bei Varicellen auf, können in exquisiter Weise durch Einreibungen des jetzt fast vergessenen Unguentum tartari stibiati erzeugt werden, und wie man aus unserm Fall ersieht, auch bei acutem Eczem auftreten. Gerade dieser Umstand spielt meiner Ansicht nach auch bei der Beurtheilung der sogenannten "generalisirten Vaccine" eine wesentliche Rolle.

Es ist hier nicht der Ort, alle Fälle die in der Literatur unter diesem Namen vorliegen, namhaft zu machen, und einzeln durchzugehen. Ich glaube behaupten zu dürfen, sie fast alle gelesen und geprüft zu haben, wobei sich ergab, dass der *grösste* Theil dieser Fälle, deren Zahl überhaupt nicht gross ist, solche Kinder betraf, die an *Eczema chronicum*, besonders des Gesichts, aber auch anderer Hautpartien litten. Aus welchem Grunde aber gerade ein Eczem die "*Generalisation*" der Impfpocken herbeiführen oder begünstigen sollte, diese Frage dürfte wohl Niemand befriedigend beantworten können. Schon dieser Umstand, das so überaus häufige Zusammentreffen mit Eczem, macht mir einen erheblichen Theil der betreffenden Fälle verdächtig, und lässt mich dieselben als acute, von der Impfung selbst unabhängige, durch Kratzen entstandene Steigerungen des schon bestehenden Hautleidens betrachten, die mit den eben besprochenen grossen und gedellten Pusteln auftraten, ganz analog dem von mir mitgetheilten Fälle, in welchem überhaupt keine Impfung stattgefunden hatte. Dass es sich in *einzelnen* Fällen auch um wirkliche Vaccinepusteln handeln kann, die durch zufälligen *Contact* der eczematösen Fläche mit dem Secret der Impfborken am Arm zu Stande gekommen sind, will ich nicht völlig in Abrede stellen. Dies kommt aber für mich gar nicht in Betracht, weil es sich in diesem Falle doch nur um die directe locale Infection des Eczems, *nicht aber* um eine von innen heraus in der Art der syphilitischen oder der acuten Exanthem auftretende Eruption handeln würde, und nur diese ist es ja, welche den Namen "generalisirte Vaccine" in Anspruch nehmen darf.

Vor einer strengen Kritik würden daher nur solche Fälle Stand halten, in denen bei *zuvor gesunder* Haut, insbesondere bei Abwesenheit

von Eczem, gleichzeitig mit der Eruption der Impfpocken am Arm, oder sehr rasch nach derselben, ein mehr oder minder reichlicher Ausbruch von *Vaccinepusteln* auf anderen Hautpartien erfolgt, ein Ausbruch der wohl nicht ohne Fieber gedacht werden kann. Ich sagte ausdrücklich "*Vaccinepusteln*," denn da auch andere Pusteln äusserlich diesen täuschend ähnlich sein *können*, z. B. Ecthyma, so müsste der Beweiss für die wirklich vaccinale Natur des Ausschlags erst erbracht werden, und dies könnte, wie ich glaube, nur durch die Verimpfung des Pustelinhalts auf noch nicht geimpfte Kinder oder junge Kälber geschehen. Dass bei den in Frage stehenden Fällen dies Experiment überhaupt und mit welchem Erfolge es gemacht worden, ist mir nicht bekannt; ich will aber gern zugeben, dass mir bei der Durchsicht der betreffenden Literatur, einzelnes entgangen sein mag. Nur bei *einem* Falle, in dem die von mir gestellte Bedingung in der That erfüllt worden ist, möchte ich desshalb verweilen.

Dieser Fall ist unter dem Titel "Vaccine généralisée à forme éruptive" von d'Espine und Icandin auf der pädiatrischen Section der deutschen Naturforscherversammlung in Düsseldorf am 20. September 1898, mitgetheilt worden.[1] Nach der Mittheilung der Krankengeschichte geben die Verfasser folgendes Resumé derselben: "Ein Mädchen von 11 Monaten zeigt am 5. Tage der Impfung mit animaler Vaccine am Arm einen Ausschlag, der vollkommen den Character von Impfbläschen trägt, zuerst auf dem Leibe und im Gesicht, dann sich vom 6–9 Tage über den ganzen Körper verbreitend, indem er durchaus gutartig bleibt. Noch bis zum 11. Tage erscheinen einige verspätete Bläschen, dann fallen die Pusteln, nachdem sie sich mit Schorf überzogen haben, fast zur gleichen Zeit mit denen der geimpften Arme, d. h. vom 19–21. Tage. Während dieser ganzen Zeit bleibt der Allgemeinzustand des Kindes vortrefflich. Die *Abimpfung der Pusteln* hat gezeigt, dass es sich auch bei dem allgemeinen Ausschlag um eine *wahre Vaccine* handelte."

Da in diesem Factum für mich die Entscheidung liegt, lasse ich die Verfasser selbst den Vorgang schildern: "Die aus den Pusteln am Bein und am Fuss gesammelte Lymphe am 8. und 9. Tage wurde am 17. Mai auf ein *Kalb* geimpft, welches vom allgemeinen Impfraum vollständig getrennt wurde. Am 23. Mai ergaben die 3 Impfstellen ein positives, aber *unkenntliches* (?) Resultat, nichtsdestoweniger zeigte eine der Pusteln die charakteristische Umwandlung, und durch die Pincette zerquetscht, lässt sie eine klare Lymphe ausfliessen. Man schabt die Pustel ab, und die Gallerte dient zur Impfung eines zweiten Kalbes am 28. Juni. Am 3. Juli ist das Resultat ein *positives* und *rein*,

[1] *Archiv f. Kinderheilk.*, Bd. xxvi, S. 367.

obgleich die vaccinalen Pusteln noch *wenig* entwickelt sind (?). Das erste Kalb wurde nach einiger Zeit einer erneuten Impfung unterzogen mit einer anerkannt activen vaccinalen Lymphe. Das Resultat blieb ergebnisslos. Das Kalb war durch seine erste Impfung immunisirt."

Man wird mir vielleicht eine zu weit getriebene Skepsis vorwerfen, wenn ich bekenne, dass mir das Resultat dieses Experiments nicht so beweiskräftig und über jedem Zweifel erhaben vorkommt, wie den Verfassern. Dazu kommt noch der Umstand, dass die Verfasser selbst anfangs auch an eine "abgeschwächte Varioloïs" dachten, weil einige Fälle derselben in einem Isolirpavillon gepflegt wurden, ein Verdacht, den sie freilich später von sich wiesen. Aber zugegeben, dass dieser nicht ganz einwandsfreie Fall in der That als "generalisirte Vaccine" zu betrachten wäre, so würde er doch meiner Ansicht erst recht die Aufforderung enthalten, die Frage durch *weitere* Experimente in etwa vorkommenden ähnlichen Fällen zu lösen. Jedenfalls würde ich den Zweck meiner Mittheilungen für erreicht halten, wenn bei der Publicirung von Beobachtungen über "generalisirte Vaccine" mit schärferer Kritik vorgegangen würde, als dies bis jetzt der Fall gewesen ist.

A CONTAGIOUS FORM OF PNEUMONIC FEVER IN CHILDREN.

By Sir HERMANN WEBER, M.D.,

Consulting Physician to the German Hospital in London, and the National Hospital for Consumption at Ventnor, Isle of Wight.

I MUST apologize for the fragmentary character of this communication, which would never have been published, had I not wished to contribute to the "Jacobi Festschrift" a short paper on a subject connected with diseases of childhood as a tribute of admiration to the man who has done such eminent work in the pathology and treatment of diseases of childhood, my fellow-student and lifelong friend, Dr. A. Jacobi of New York.

In 1869 I observed an epidemic of moderate pyrexia with lobar pneumonia of a mild character and a typical course. The disease seemed to have originated at a boys' school in Essex, and to have spread only amongst children who had come into intimate contact with boys from that school. I am not quite satisfied with the name, but I do not know a better one. We call scarlet fever an epidemic disease with a scarlet rash; enteric fever an infectious fever with enteric ulcers; so I venture to call pneumonia fever or pneumonic fever an infectious fever the prominent manifestation of which is a lobar pneumonia. I am aware, however, that there are different kinds of pneumonic fever, caused by different microbes, and requiring distinguishing names.

Case 1.—A. K., ten and a half years old. On July 31, 1869, I was called to Highbury New Park to see a boy who had been sent home from a school in Essex, where various cases of illness had occurred amongst the boys of the school. A. K. had been ill for three or rather four days with fever and diarrhœa. On the 28th he had shivering and headache, and afterwards a moderate amount of diarrhœa. He had a slight cough, and over the lower part of the left side from the scapula downwards there was some dulness on percussion, bronchial breathing, and crepitant rhonchus.

July 31st. At 9 A.M. T. 103° F. P. 96. R. 30. *Treatment:* Rest in bed; milk and limewater.

At 6 P.M. T. 103.6° F. P. 104. R. 32.

Aug. 1st. 8 A.M. T. 102.5° F. P. 96. Diarrhœa almost gone ; lung symptoms the same.
5 P.M. T. 100.4° F. P. 90. Perspiration.
" 2d. 9 A.M. T. 97.5° F. P. 62. R. 20. Boy feels almost well; the local lung symptoms slightly diminished.
" 3d. 9 A.M. T. 96.2° F. P. 58, very irregular. Dulness much diminished, bronchial breathing disappeared, coarse rhonchi.
" 8th. 5 P.M. T. 98.0° F. P. 64. Dulness and rhonchi almost disappeared, but occasional friction sounds. General condition normal, but is rather pale and weak.

He is allowed to go to friends in the neighborhood of Epsom on Aug. 9th.

On August 20th he returns, feels very weak, and has violent pain in both legs. There is slight pyrexia. T. 99.2°. P. 85.

Under the influence of rest, good food, six ounces of port wine, and three grains of quinine every day the pain in the legs almost disappeared in the course of a fortnight, and after some further weeks at Margate the general health and strength became satisfactory.

Case 2.—W. K., nine years old, a brother of A. K., with whom he had often been since August 1st, fell ill on August 14th with headache and nausea, without actual sickness or diarrhœa, and without any lung symptoms.

Aug. 14th. 8 P.M. T. 102° F. P. 105. *Treatment:* Rest ; light diet and effervescent citrate of potash.
" 15th. 9 A.M. T. 102.8° F. P. 98.
7 P.M. T. 103.6° F. P. 110.
There is occasional slight cough, but there are no well marked local symptoms.
" 16th. 8 A.M. T. 103.5 . P. 102.
6 P.M. T. 104 . P. 112.
Distinct dulness on percussion over the lower part of the right side, some crepitation, and occasional pleuritic rubbing ; scarcely any cough or pain.
" 17th. 8 A.M. T. 103°. P. 104.
8 P.M. T. 103.4°. P. 108.
The local symptoms in the right lung well marked. No cough. No pain.
" 18th. 9 A.M. T. 101° F. P. 96.
6 P.M. T. 98.8°. P. 74.
There is still slight dulness and coarser rhonchi over the lower part of the right side.
" 19th. 8 A.M. T. 96.2° F. P. 65.
" 20th. 7 P.M. T. 98.2° F. P. 68.
The boy made a good convalescence and had no sequelæ calling for remarks.

Case 3.—K. K., seven and a half years old, a healthy child, sister of Cases 1 and 2, with whom she frequently played during their illness, remained well until 15th August, when she complained of headache, heat, and diarrhœa.

Aug. 15th. 7 P.M. T. 101° F. P. 104. *Treatment:* Rest ; milk and limewater.
" 16th. 8 A.M. T. 102° F. P. 106.
6 P.M. T. 103.8°. P. 112.
Very slight dulness over the lower posterior part of the left side, and diminished respiratory sounds without rhonchi.
" 17th. 8 A.M. T. 104.2° F. P. 114. R. 38.
8 P.M. T. 104.6° F. P. —. R. 36.

Aug. 17th. 8 P.M. Dulness on left side increased, and crepitant rhonchi. Very little cough. Diarrhœa has ceased.

" 18th. 9 A.M. T. 103.2° F. P. 108. R. 36.

6 P.M. T. 103° F. R. 32.

More crepitant rhonchi. Perspires freely; is very drowsy.

" 19th. 8 A.M. T. 101° F. P. 78. R. 28.

7 P.M. T. 98.8° F. P. 68.

The local condition of the lung scarcely changed, but towards morning of this day (19th) the child became delirious. I found her at 8 A.M. bathed in perspiration and crying, saying that her rabbits had been killed. This state lasted the whole day, until late at night she fell into a sound sleep which continued more than fourteen hours.

The lung symptoms disappeared within a few days but the child remained very weak, the pulse was for more than a week very irregular and below 65, the temperature under 97.5° F. Later on the progress was satisfactory.

CASE 4.—A younger sister, five years old, had somewhat later, viz., 22d to 24th August, a slight attack of indisposition, consisting in headache, want of appetite, increased thirst, and a slight rise of temperature without any pulmonary symptoms, and was quite well after three or four days.

CASE 5.—On the 23d of August I was called to Epsom into the house where A. K. (Case 1) was on a visit since the 9th of the month. M. B., a girl, nine years old, who had played much with A. K., had on the evening of the 22d a slight rigor with headache and pain in the limbs.

When I saw her at 4 P.M. she was very thirsty; had a little cough. T. 103° F. P. 110.

There was slight dulness and crepitant rhonchus over the lower part of the left side; scarcely any cough.

Aug. 24th. 6 P.M. T. 103.2.° P. 118. R. 40.

The pulmonary symptoms more marked.

" 25th. 5 P.M. T. 97.5°. P. 64; very irregular.

Local pulmonary symptoms about the same, excepting that the rhonchus was coarser and moister.

On September 4th there were no local symptoms left; she was convalescent, but rather weak.

On September 20th I saw M. B. again, because she had had an attack of syncope, had great weakness in the limbs, and could not play or do anything.

I found her very pale and anæmic with a weak, irregular pulse of 55 to 60; a dilated heart with anæmic basal systolic bruit. T. only 96.5° F.

Treatment: Perfect rest on an open veranda from morning till night; frequent slight meals with port wine and Blaud's pills. The recovery was slow, but at the end of October she could be moved to Eastbourne, where she was constantly in a Bath chair, and she became perfectly well before Christmas.

CASE 6.—L. B., aged ten and a half, brother of M. B. and friend of Case 1, had on August 22d diarrhœa, vomiting, and headache.

Aug. 23d. 4 P.M. T. 102°. P. 102.

Slight cough, and occasional pain in the left side. Some crepitant rhonchi and occasional friction sound over the lower part with moderate dulness from the middle of the scapula downwards.

Treatment: Rest; milk and limewater.

" 24th. 5.30 P.M. T. 103.2° F. P. 104.

Pulmonary symptoms more marked.

" 25th. 5 P.M. T. 103.8° F. P. 106.

General condition better; more food.

" 26th. 9 A.M. T. 101.8°. P. 88.

Aug. 27th. 9 A.M. T. 97.2° P. 66; irregular.
Patient feels weak, but otherwise well. The lower part of the left lung is clearing up.
From this date the recovery proceeded without interruption and without sequelæ.

Three other children of this family were sent away from the house to the seaside on August 23d. One of them, a boy of six, has been ill, while there, between the 6th and 10th of September, with what the doctor called slight congestion of the lungs.

Case 7.—C. T., a boy aged ten, who lived close to the house of Cases 5 and 6, and had played on the 9th and 10th of August with Case 1, became ill on the 22d of August with pain in the limbs and fever. The medical man who attended him thought first of rheumatic fever and gave him lemon juice. On the 24th, however, the boy manifested all the symptoms of pneumonia in the lower part of the right lung and entered convalescence on the 28th.

A younger brother of Case 7 is said to have been taken ill at Margate on the 5th or 6th of September in a similar way as his brother, and to have recovered after six or seven days.

On making enquiries at the school in Essex from which A. K. (Case 1) had returned ill on July 30th, I learnt that since the beginning of July many cases of illness had occurred but that none of them had been very severe, and that the symptoms had not been the same in the different cases—in some boys diarrhœa had been the prominent feature, in others rheumatic pains with fever;—that there had not been any decided lung symptoms calling for examination of the lungs, but that in two boys slight congestion of the lower lobes of the lungs had been discovered. It had been assumed that the closets and drains had been out of order. During the holidays, therefore, a thorough alteration of the drainage and of the arrangements of the house was made, and no illness occurred at the school after the return of the boys.

I further heard that in the beginning of the holidays several of the brothers and sisters of the boys returned from school were taken ill, but that these illnesses were not regarded as connected with the illnesses of the boys coming from school, and that they were of a mild character and of short duration. They were called congestion of the lungs, feverish bilious attacks, and febrile rheumatism, and attributed to chills and mistakes in diet.

This peculiar contagious affection excited my interest in a high degree at the time of observation, and it had been my intention to publish my notes, but first my holidays intervened, and on my return urgent practical work in another field of our profession prevented my searching the literature for a similar epidemic. I also felt that my observations were imperfect and hoped to meet with another epidemic allowing me to make them more accurate and to gain a better insight into the nature of the disease; but this opportunity never occurred. Others have seen, as well as myself, that common lobar pneumonia can be communicated from one person to another, and quite lately

I observed in a severe case of pneumonia that the nurse and a sister of the patient were attacked with the disease. I have also at different times seen ordinary pneumonia occurring in an epidemic form, and several epidemics have been described. These epidemics of ordinary lobar pneumonia are, however, decidedly different from the epidemic which is described in this paper, since the former shows a much shorter stage of incubation, occurs especially in adults, and is usually fatal in a large percentage of cases. The cases of the epidemic here described had all the characters of an acute, very contagious disease, like measles, scarlet fever, mumps, varicella. Imperfect though my observations are, we can recognize a distinct stage of incubation from eleven to thirteen days; a stage of manifestation of disease, lasting generally four to six days, with varying general symptoms, but regular localization in the lower parts of the lungs, and a rapid decline. We have also in Case 4 a very mild abortive form, such as we see not rarely in scarlet fever, whooping-cough, measles, and other infectious diseases. We have further cases with sequelæ, such as a kind of peripheric neuritis in Case 1, and great weakness of the heart with anæmia in Case 2. We have further a case of that peculiar form of delirium of the decline in febrile diseases, such as we occasionally meet with in ordinary lobar pneumonia, in measles, scarlet fever, and other acute infectious diseases (*Med.-Chir. Transactions*, vol. xlviii., p. 199, "On Delirium or Acute Insanity, during the Decline of Acute Diseases, especially the Delirium of Collapse").

I am not acquainted with a description of a similar affection, although such a description may exist and may have escaped my notice, in which case I would beg indulgence for my ignorance. I am inclined to think that through peculiar circumstances existing in the school a variety of a microbe may have been produced which caused this variety of a contagious affection.

Although the occurrence of the disease in this epidemic was confined to children, it does not follow that it may not occur also amongst adults, as we observe it in mumps, measles, etc. The mildness which characterized these cases need likewise not always exist.

My son, Dr. Parkes Weber, has directed my attention to epidemics of jaundice in children, of which several descriptions have been given by different observers, and quite lately by Friquet in *Presse Médicale*, July, 1899 (Epitome of *British Medical Journal*, 26th August, 1899), and more fully by A. A. Kissell in *Jahrbuch d. Kinderkrankheiten*, vol. xlviii. ("Ueber infectiösen Icterus bei Kindern"). Similar epidemics have been described in adults, and it is quite possible that epidemics of the disease here described may also occur occasionally amongst adults.

EIN BEITRAG ZUR KENNTNISS DER MEDULLÄREN (MYELOGENEN) LEUKAEMIE IM KINDESALTER.

VON ADOLF BAGINSKY.

IN A. Jacobi's lehrreichem und allen, mit der Kinderheilkunde sich befleissigenden Praktikern, wohl bekanntem und von ihnen werthgeschätztem Buche: *Therapeutics of Infancy and Childhood* ist mir das Capitel: "Diseases of the Blood and Constitution" eines der schätzens werthesten, neben den vielen anderen erschienen, und so glaube ich nicht übel daran zu thun, wenn ich einen bemerkenswerthen Fall von Leukaemie zum Gegenstande einer kurzen klinischen Mittheilung mache, wenngleich dieser Fall nicht zu Ende beobachtet wurde, sondern ungeheilt unser Krankenhaus verliess.

Am 6. October 1898 wurde im Kaiser und Kaiserin Friedrich Kinderkrankenhause der 9jährige Knabe Arthur Zimmermann auf der chirurgischen Abtheilung aufgenommen.

Anamnese. — Der Vater des Knaben ist mit 39 Jahren an einem Herzleiden gestorben, die Mutter ist gesund und lebt noch.— Der Knabe hat im zweiten Jahre Masern gehabt und Keuchhusten.

Im vergangenen Sommer hat derselbe eine Lungenentzündung überstanden. Im August des Jahres 1898 begann der Leib zu schwellen, und nahm langsam zu bis zu dem Umfange, den er augenblicklich hat. Der Appetit nahm ab. Stuhlgang wurde unregelmässig und oft angehalten. Es trat zeitweilig Nasenbluten ein, und vor vier Wochen zeigte sich etwas Schwellung der unteren Extremitäten. Die Hautfarbe wurde auffallend bleich und der Knabe wurde immer elender.

Stat. präsens: Elender bleicher Knabe, schlechtes Fettpolster, dürftige Muskulatur; flacher Thorax. Haut ohne Exanthem, ohne Narben. Keine bemerkenswerthe Schwellung der Lymphdrüsen. Tiefe Bleiche aller Schleimhäute.

Die Lungen sind intact. Die Respiration rein vesiculär. Die Herzdämpfung beginnt in der Höhe der 3. Rippe. Spitzenstoss im 5. Intercostalraum, ein wenig ausserhalb der Mammillarlinie. Reine Herztöne. Normale Herzaction.

Die Leber überragt nur wenig den unteren Rippenrand; ist kaum zu palpiren.

In der Milzgegend präsentirt sich ein mächtiger Tumor mit glatter Oberfläche und scharfem, mit Einkerbung versehenem Rande. Derselbe erstreckt sich nach vorn bis zur Mittellinie. Nach unten reicht der Tumor bis in's Becken und tritt nahezu bis an das Ligam. Poupartii sinistr. heran.— Der Umfang des Abdomen ist beträchtlich; 67 Cm. unterhalb des Rippenbogens, 61 Cm. um den Nabel gemessen. Keine freie Flüssigkeit in der Bauchhöhle. Harn frei von Albumen und anderan fremden Bestandtheile.— Sinnesorgane intact.— Keinerlei spontane oder Druckschmerzen.— Die erste Blutuntersuchung giebt

spez. Gewicht des Blutes 1049
Haemoglobingehalt.....35 %
rothe Blutkörperchen..1,640,000 im cb.cm.
weisse " .. 410,000 " "
Verhältniss der rothen und weissen wie 1 : 4.

Der vorläufige mikroskopische Blutbefund zeigt eine enorme Vermehrung der mononucleären Leucocyten, auch der Eosinophilen und Mastzellen. Reichliche kernhaltige rothe Blutkörperchen. Noch am Tage der Aufnahme reichliches Nasenbluten. Temp. 37,5, Puls 120. Ordination: Levicowasser.

In den nächsten Tagen das gleiche Verhalten; zeitweilig dyspnoeisches Athmen. Starke Schweisse. Als kein Object chirurgischer Behandlung wurde der Knabe der inneren Abtheilung überwiesen.

Man constatirte hierselbst in Ergänzung des gegebenen Status noch Folgendes: Neben der hochgradigen Blässe der Haut, markirten sich starke bläuliche Venenzüge in der Haut der Brust und des Bauches. Die Haut ist zeitweilig mit profusem Schweiss bedeckt. Die Athmung ist beschleunigt, dyspnoeisch und zeitweilig orthopnœisch.—Ueber dem etwas fassförmig gestalteten Thorax hört man jetzt weithin schnurren, auch ist die Athmung wenig laut.— Die Herzaction ist leicht arythmisch und etwas beschleunigt.— Die Leber jetzt deutlicher palpirbar als früher, reicht fast bis zur Nabellinie; dieselbe ist scharfrandig, glatt an der Oberfläche.— Der Harn ist reich an Uraten, enthält nur ganz wenig Phosphate und zeigt geringe Trübung bei der Eiweissprobe.

Temp. 39° C., Puls 148, Rp. 48.

Im weiteren Verlaufe war das Befinden des Kranken ausserordentlichen Schwankungen unterworfen. Am 18. November: Schwere Dyspnoea und dementsprechend starke Füllung der Hautvenen und allgemeine Cyanose. Schlaflosigkeit. Anorexie. Temp. zwischen 39° C. Abends und 37,2 Morgens.

Die Untersuchung des Blutes ergiebt

spez. Gewicht.....1046
Haemoglobingehalt.....30–35 % Fleischl.
rothe Blutkörperchen.....2,133,000
weisse " 986,000
Verhältniss der rothen zu weissen wie 1 : 2, 2.

Bei der Blutentnahme aus der Fingerkuppe tritt ein dünnes, trübes, lackfarbenes Blut zu Tage. Dasselbe fliesst anfangs rasch, bald aber restirt der Abfluss. Die freie Vertheilung des Blutes auf dem bereit gehaltenen Deckgläschen ist äusserst schwierig. Das Blut ist von eigenthümlicher zäher Klebrigkeit.

An den trocken mittels Ehrlich's Triacidgemisch gefärbten Blutpräparaten ist zunächst das gesammte Blutbild ein höchst eigenthümliches und von dem Aussehen normalen Blutes durchaus abweichendes.

Das Blut präsentirt sich im Grossen als ein auf der Fläche ausgeleertes Gemisch von mächtigen mononucleären und polynucleären Leucocytenformen, zwischen denen die rothen Blutkörperchen sich mit einander verklebt oder auch einzeln liegend, nahezu verlieren. Grobkörnige Leucocytenformen, ein- oder mehrkörnig, tauchen aus der Masse der monotonen das Gesichtsfeld beherrschenden grossen Leucocytenformen auf, dazwischen ausserdem zerstreute Körnerhäufchen von zerstreuten gekrönten Blutzellen, zwergförmige kleine mononucleären Leucocyten und endlich einzelne ein- oder mehrkörnige mit scharf umgrenztem rundem Kern versehene rothe Blutkörperchen.— Man würde vergebens sich bemühen, das ganze Bild mit demjenigen normalen Blutes auch nur in Analogie zu bringen.

Im Einzelnen ergiebt die Untersuchung Folgendes:

Die rothen Blutkörperchen sind im Ganzen nur gewöhnlicher und normaler Form, dagegen zeigen dieselben in den Grössenverhältnissen die äussersten Verschiedenheiten. Ganz grosse runde Scheiben wechseln in auffallendster Weise mit kleineren ab. Wo sie zwischen den Leucocytenmassen wie eingeschoben liegen, erkennt man, dass sie zu Geldrollen an einander geklebt sind. Einzeln liegende zeigen spärlich zwar, aber deutlich auch hervortretende längliche und Keulen-Formen (Poikilocyten). Die Farbe der Blutkörperchen ist leuchtend roth. Mitten in den an sich normalen Gebilden findet man mit grossem, schönem, heftig dunkelblauem Kerne versehene Körperchen; einzelne von ihnen enthalten auch zwei kreisrunde Kerne.

Es handelt sich demnach schon bei den rothen Blutkörperchen um die Anwesenheit anormaler Bildungen.

Unter den Leucocytengebilden sind die meistens vorherrschenden mächtige, grosse, mit einheitlichem, unregelmässig begrenztem grossem Kerne versehene Zellen. Der Kern färbt sich im Triacidgemisch

dunkel, während die protoplasmatische Zellmässe eben nur Andeutungen feiner Körnung zeigt. Vereinzelt findet man allerdings an den Rändern auch gröbere Granulirung. Dieselben sind an Zahl so beträchtlich, dass sie die übrigen nahezu unterdrücken. Unter 97 ausgezählten Zellen finden sich 55 dieser Gebilde = 56.7 %.

Demnächst ist die Zahl der polynucleären Zellen auffällig. Die Kerne sind von der mannigfachsten Gestalt und Zahl, vielfach in Keulenform, Sackform, eingeschnürt wurstförmig und ebenso mitunter bis zu sechs in einer Zelle.—Die Granula erscheinen feinkörnig, blasser als die dunkel und markig sich färbenden Kerne. Ihre Zahl beträgt unter 97 Zellen 35 = 36.1 %.

Eosinophile Zellen finden sich mit grober Körnelung und mononucleären oder polynucleären Kernen. Dieselben lassen neben dem graublau oder gräulich gefärbten Kern die dicken und scharfumgrenzten Granula leuchtend roth hervortreten. Unter 97 Zellen sind 4 derselben erkenntlich = 4.1 %.

Ausser diesen Zellen beobachtet man in mit basischen Farbstoffen gefärbten Präparaten auch einzelne basophile Zellen mit grossen einheitlichen Kernen.

Endlich kleine, einkernige, sich dunkelfärbende Leucocyten von kleiner Gestalt und geringem Umfange (Zwergformen).

Im Ganzen handelt es sich hiernach um eine enorme Vermehrung mononucleären Leucocyten, "Ehrlich's Myelocyten," in erster Reihe und der ganze Krankheitsfall muss in die Gruppe der myelogenen Leukaemien eingereiht werden.

Das Blutbild, im weiteren Verlaufe mehrfach untersucht, änderte sich nicht wesentlich. In der Zahl der eosinophilen Zellen und eben so auch in der der kernhaltigen rothen Blutkörperchen schienen Schwankungen aufzutreten, die indess bei Auszählungen nicht absolut sichere, sondern in verschiedenen Präparaten verschiedene Zahlen ergaben.

Auch der gesammte Krankheitsverlauf bot mannigfache und seltsame Schwankungen. Tage relativer Euphorie wechselten mit Tagen schwersten Leidens. Während der Knabe die Nächte bisher ruhelos verbracht hatte und nur mittelst Morphium zum Schlaf kommen konnte, liess am 24. Dyspnoea, Schmerzhaftigkeit des Abdomens so nach, dass der Knabe sich leidlich wohl fühlte. Der Harn zeigte einen geringen Albumingehalt. Die Temperatur nahezu normal = 37.4°–38° C. schwankend. Puls 132, Rp. 48. Die Behandlung bestand im Wesentlichen in roborirender Diät und Gebrauch kleiner Mengen von Levicoquelle.

Am 10. December zählte man

rothe Blutkörperchen	1,978,880	rothe und weisse 1:2, 2
weisse "	884,210	

Haemoglobingehalt 26–30 % Fl.

Am 11. December folgte ohne nachweisbare Ursache eine Temperatursteigerung bis 38.5, die sich des Weiteren fortsetzte und am 13. in der Nachmittagsperiode bis 40.4 anstieg.—Das Allgemeinbefinden schlecht. Grosse Abgeschlagenheit. Dick belegte Zunge. Anaemie. Leichte Pharyngitis. Oefteres Rasseln über den ganzen Thorax. Starke Cyanose. Die Venen traten an der Haut des Thorax stark gefüllt heraus. Puls elend, 162, Rp. 48. Die Fieberepisode dauerte mit Schwankungen bis zum 18. December.—Erneute Blutuntersuchung ergab am 15. December

rothe Blutkörperchen 2,000,000
weisse " 980,000 } 1:2, 5
Haemoglobingehalt, 25 % Fl.

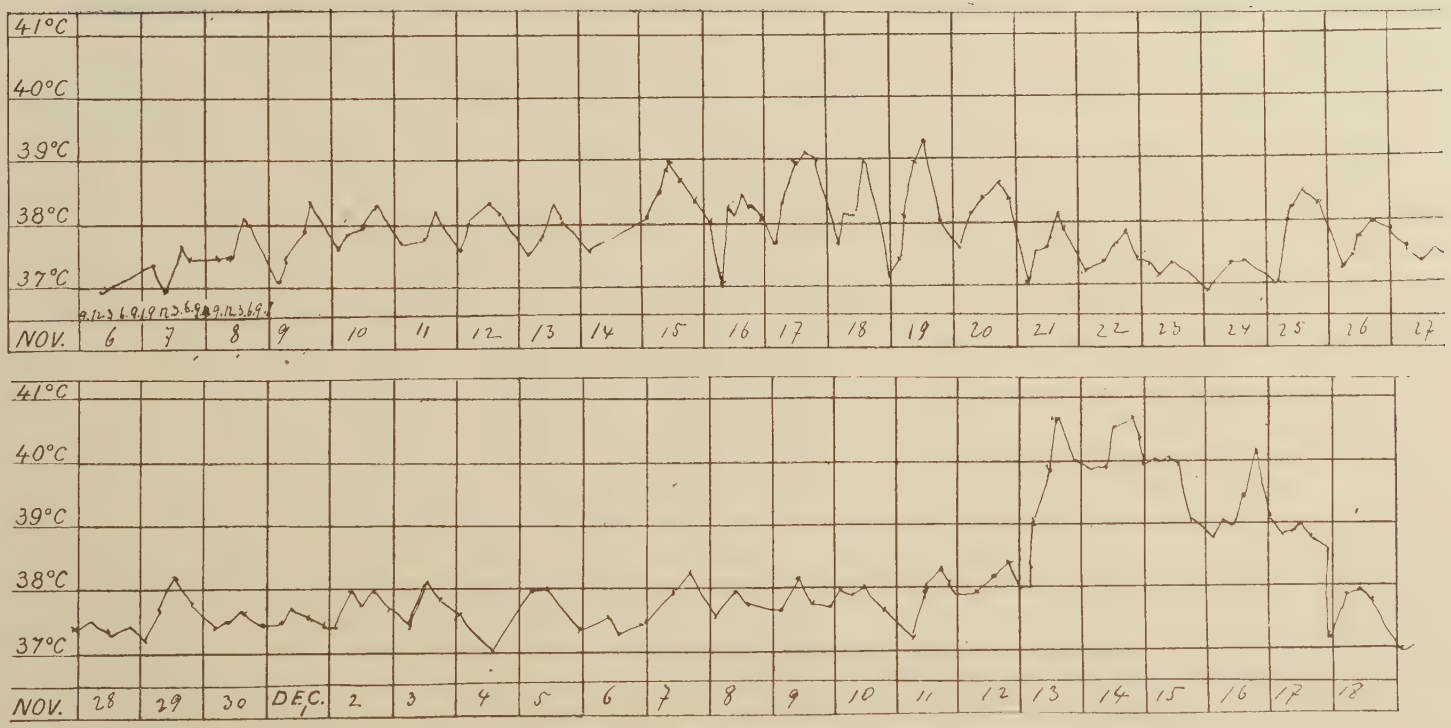
Im mikroskopischen Blutbilde sind die rothen Blutkörperchen wesentlich mehr als früher in ihrer Gestalt alterirt. Grosse blasse rothe Blutzellen wechseln mit kleinen und kleinsten Formen. Die Gestalt ist vielfach verändert, keulenförmig, aber unregelmässig.—Zahlreich finden sich kernhaltige rothe Blutkörperchen. Von den Leucocyten überwiegen die grossen mononucleären Formen mit unregelmässigem, oft fast die ganze Zelle einnehmenden grossen Kerne; derselbe enthält oft kleine hellere, rundliche Einschlüsse. Unter 120 Leucocytenformen sind

95 mononucleäre (Myelocyten) = 79.1 %
15 polynucleäre = 12.3 %
6 eosinophile Zellen = 5 %

Der Knabe musste, nachdem er am 19. December wieder völlig entfiebert war, auf Wunsch der Eltern entlassen werden und befand sich bei der Entlassung in folgendem Zustande: Derselbe sieht tief bleich und elend aus; indess befindet sich derselbe immerhin so leidlich, dass er das Bett verlassen und umhergehen kann. Leichte Cyanose der Lippen. Unbedeutende Dyspnoea. Ueber den Lungen überall vesiculäres Athmen, kein Rasseln. Normale Percussionsverhältnisse.— Die Herzdämpfung beginnt an der 3. Rippe, überragt den linken Sternalrand nur um ein Weniges. Spitzenstoss im 5. Intercostalraum. Herztöne ein wenig dumpf.

Auf dem Abdomen weithin ausgedehnte blaue Venen. Leibumfang in Nabelhöhe 64.5 Cm. Zwischen Nabel und Processus xiphoideus 72 Cm. Leber gross, glatt an der Oberfläche, überragt den Rippenbogen um 8 Cm. Die Milz ist colossal, ein harter Tumor, mit tiefen Einkerbungen am Rande. Die grösste Breite der leicht palpirbaren Milz ist 10 Cm., die grösste Länge 70 Cm. Das Abdomen, welches früher sehr schmerzhaft war, ist schmerzlos. Keinerlei Ansammlung von freier Flüssigkeit im Abdomen.

Puls 144. Rp. 48. Temp. 37.0° C.



TEMPERATUR KURVE—FALL VON MEDULLÄRER (MYELOG.) LEUKAEMIE.

Der gesammte Krankheitsfall präsentirt sich nach der Art des von ihm dargestellten Blutbildes als eine vom Knochenmark ausgehende echte myelogene Leukaemie im Sinne der von Ehrlich[1] neuerdings gegebenen Eintheilung, bei welcher der mächtige Milztumor mehr passiv und vielleicht nur insofern betheiligt ist, als hier ein Anpassen von Myeloidgeweben stattgefunden haben. Derselbe zeichnet sich aus einmal durch den langsamen, schleichenden, von verschiedenartigstem Verhalten des Wohlbefindens begleiteten Verlauf, durch die tiefe Alteration der Respiration, die sich bis zur schweren Cyanose und selbst intensiver Orthopnoea steigern konnte; zeitweilig war der Befund derartig, dass man den Eintritt des suffucatorischen Todes befürchten musste.—Auffallende nervöse Störungen waren, abgesehen von der hin und wieder eintretenden grossen Unruhe, die auch den Schlaf nicht aufkommen liess, nicht vorhanden. Schwere Abgeschlagenheit bis zum Eintritt comatösen Zustandes war allenfalls während der fieberhaften Periode vorhanden. Sonst aber Sensibilität und Mobilität ebensowenig gestört, wie das Sensorium. Höchst auffällig sind die in mehreren Attaquen auftretenden Fieberbewegungen, für welche ein zeigender Grund nicht vorhanden oder aufzufinden war.

Gelegentlich einer Studie über acute Leukaemie erläutert A. Fraenkel,[2] dass bei zwei von ihm beobachteten Fällen mit dem Einsetzen einer septischen Infection und erheblicher Fiebertemperaturen eine sehr erhebliche Verminderung der Leucocytenzahl verbunden gewesen sei und bezieht dieselbe auf den Vorgang eines wirklichen Leucocytenfalles, auf Leucocytolyse. Nach den an unserem Falle gemachten Zählungen war während der Fieberperiode in der Zeit vom 11. bis zum 17. December von einer derartigen Leucocytolyse nichts zu bemerken. Es veränderte sich ebenso wenig die Zahl der Leucocyten in toto, wie auch das Blutbild in Beziehung auf das Verhältniss der mononucleären zu den polynucleären Elementen sich nicht in irgend auffälligem Maasse verschob.

Der Krankheitsfall gab uns Anlass, den von früheren Autoren geschilderten und in der Pathologie der Leukaemie bereits seit langer Zeit bekannten Veränderungen der Harnbeschaffenheit nachzuforschen.

In der Zeit der Fieberperiode vom 13. bis zum 16. December, während Patient sich bei flüssiger Fieberdiät (Milch, Bouillon, Purofleischsaft) befand, gestaltete sich die Harnausscheidung folgendermassen:

Harnmenge Dez. 13. — 1300 Ccm.	Harnmenge Dez. 15. — 1650 Ccm.
" 14. — 1950 "	" 16. — 1280 "

[1] P. Ehrlich und A. Lazarus: *Die Anaemie*, Bd. viii, Th. i, in dem von Nothnagel herausgegebenen Werke der *Speciellen Pathologie und Therapie*, pag. 116.

[2] A. Fraenkel: *Deutsche med. Wochenschrift*, 1895, No. 42.

In dem Harn wurde nach Kjeldahl die Gesammtstickstoffmenge bestimmt

Total N. Dez. 13. = 12.88 g. Total N. Dez. 15. = 15.85 g.
" 14. = 16.17 " " 16. = 12.43 "

Die Harnsäuremenge wurde nach Ludwig-Salkowsky durch Fallung mit Magnesia-Silbermischung, Wägung der zum constanten Gewicht getrockneten Säure aufgezogenen Filter bestimmt.

Harnsäure Dez. 13. = 1.30 g. Harnsäure Dez. 15. = 1.50 g.
" 14. = 1.45 " " 16. = 1.23 "

Das Verhältniss des Harnsäurestickstoffes zum Totalstickstoff stellt sich danach folgendermassen:

Dez. 13. = 1 : 30 Dez. 15. = 1 : 32
" 14. = 1 : 32 " 16. = 1 : 30

Legt man das durchschnittliche Normalverhältniss des Harnsäurestickstoffs zum Totalstickstoff wie 1 : 50 zu Grunde, so erkennt man auch in unserem Falle wieder eine erhebliche Vermehrung der Harnsäure, die, möge man auch die Ursache derselben noch vielfach discutiren, nach den Untersuchungen von Horbaczewski und späteren zahlreichen Forschern, wohl kaum anders, als auf das Verhalten der Leucocyten bei der Krankheit zu beziehen haben wird.

Auch der Ammoniakstickstoff wurde, und zwar nach der bekannten Methode von Schlösing, bestimmt und zeigte folgende Mengen:

Dez. 13. = 1.23 Dez. 15. = 1.28
" 14. = 1.25 " 16. = 1.27

Danach erscheint auch das Verhältniss des N. 713 Stickstoff zum Gesammtstickstoff vermehrt.

Während hierbei die von früher schon bekannten Thatsachen neue Bestätigung fanden, konnten wir auf der anderen Seite das Vorkommen von anderen fremdartigen Bestandtheilen im Harn nicht bestätigen.— Die Untersuchungen auf Gallenfarbstoff, Gallensäure, Albumose, Aceton, Diacetessigsäure, Oxybuttersäure, Milchsäure ergaben stets ein negatives Resultat.

Ueber die Behandlung des Patienten ist wenig zu sagen. Wir haben weder mit guter roborirender Diät und besten hygienischen Einwirkungen noch mit dem Gebrauch der Arsen-Eisenquellen von Levico einen nennenswerthen Erfolg zu erzielen vermocht. Während der Fieberperiode und der während derselben bestehenden Dyspnoea haben Sauerstoff-Inhalationen und kühle Einpackungen dem Patienten wohlgethan. Im Ganzen verliess derselbe zwar einigermassen gebessert, indess doch mit nicht verändertem Leiden das Krankenhaus.

SUR L' EMPLOI DE L' ASAPROL DANS LE TRAITEMENT DE LA COQUELUCHE.

PAR LE DOCTEUR MONCORVO,

Professeur de clinique pédiatrique à Rio de Janeiro ; Membre correspondant de l'Académie de Médecine de Paris.

DÈS 1883, j'ai signalé l' efficacité remarquable des badigeonnages à l'entrée de l'arbre aérien avec une solution de résorcine, dans le traitement de la coqueluche, démontrant en même temps que le germe de cette affection trouve dans le larynx son siège d'élection. Or, ces premiers succès ne tardèrent point à être contrôlés par un grand nombre de cliniciens, en Europe et en Amérique, et je me crois aujourd'hui autorisé à dire que nous avons dans ce precieux agent antiseptique l'un des moyens les plus actifs et les plus sûrs contre le micro-organisme de la coqueluche. Sans vouloir m'occuper ici de la pathogénie de cette affection, je dirai que le germe que j'ai décrit comme étant la cause du mal, cultivé, et inoculé à des animaux, leur a constamment communiqué la coqueluche. En 1891, j'ai confié à Moncorvo fils, chargé des travaux bactériologiques dans mon laboratoire et aujourd'hui mon chef de clinique, le soin de poursuivre mes recherches, ce qu'il a fait avec la technique la plus rigoureuse, en étudiant le pouvoir bactéricide des différents agents antiseptiques, soit directement sur le germe, soit sur leurs milieux de culture. Or, il ressort de ses recherches, que le sublimé, la résorcine, l'acide citrique et le benzonaphtol ont été les seuls antiseptiques capables de détruire promptement le germe, ou d'en empêcher le développement dans les cultures. Les données bactériologiques ont confirmé les suites heureuses de l'emploi de la résorcine. Plus tard, essayant, dans les cliniques, l'action de l'acide citrique selon la méthode adoptée pour la résorcine, nous avons également réussi dans un certain nombre de cas. Le benzonaphtol ou le naphtol-B ne se prêtent pas à cet usage, à cause de leur insolubilité aussi bien qu'en raison de leur action quelque peu irritante à la dose à employer. Le dérivé soluble du naphtol-B, c'est-à-dire l'asaprol, introduit dans la thérapeutique par Dujardin-Beaumetz et

Stakler, ne pouvait donc manquer d'attirer mon attention, et je me décidai à en étudier l'action dans un certain nombre de cas de coqueluche.

Obs. I.—Garçon de 15 mois, admis à l'hôpital pour une fièvre traitée d'ailleurs avec succès par l'asaprol donné à l'intérieur. Pendant son séjour, l'enfant gagne la coqueluche. Badigeonnages avec une solution d'asaprol à 1 pour 100, pratiques toutes les deux heures. Au bout de trois jours l'enfant n'avait plus de quintes ni de manifestations malariennes.

Obs. II.—Fillette de 36 jours, syphilitique. Coqueluche reconnue depuis 3 jours, contractée d'autres enfants. Quintes provoquées par la compression du cartilage thyroïde; également traitée avec succès par les badigeonnages asaprolés.

Obs. III.—Garçon de 6 mois, entré à l'hôpital le 18 septembre 1894. Coqueluche datant de 15 jours. Traitement commencé le 22 septembre; disparition des quintes le 27.

Obs. IV.—Fillette de 7 mois, entrée le 12 septembre 1894. Coqueluche remontant à 15 jours; 20 quintes par jours avec vomissements plus fréquents la nuit. Badigeonnages. Le 15 elle n'a plus que 10 quintes, le 20 la reprise a cessé, le sommeil revient.

Obs. V.—Fillette de 11 mois, syphilitique. Le 24 août quintes; commencement des badigeonnages. Le 3 septembre, 3 jours après son entrée, la coqueluche était enrayée avant d'avoir atteint la période convulsive.

Obs. VI.—Garçon de 5 mois, admis le 12 septembre pour accidents palustres, gagne la coqueluche qui se manifeste, le 22 octobre, par 20 quintes par jour; on fait le traitement. Le 25 les quintes ne sont déjà plus suivies de reprise et, le 5 novembre, il n'y en a plus.

Obs. VII.—Fillette d'un an et demi, entrée le 11 octobre 1886. Coqueluche datant de 15 jours, en pleine période convulsive; 20 quintes violentes avec vomissements par 24 heures. Après 3 jours, le 13 octobre, les quintes ont diminué; le 23 la coqueluche peut être considérée comme guérie.

Obs. VIII.—Garçon de 2 ans et demi, entré le 7 décembre; coqueluche reconnue depuis 4 jours. Une quinte avec reprise par heure. Guérison en une semaine.

Obs. IX.—Fillette de 3 ans, vue le 23 octobre pour la première fois. La coqueluche remonte à 4 semaines. Quintes intenses, ulcération du frein de la langue. Dès le 22 octobre, après 2 jours de traitement, amélioration; le 26 il n'y a plus de quintes.

Obs. X.—Garçon de 5 ans, tuberculeux, entré le 5 juin 1894; gagne la coqueluche, qui est très nette, le 29 septembre. Le 30 les quintes

diminuent. Le 1er octobre, la compression du cartilage thyroïde n'arrive pas à les provoquer. Il reste une adénopathie trachéo-bronchique pour laquelle on fera un traitement approprié.

Obs. XI.—Fillette de 6 ans, syphilitique, paludique ; signes de tuberculose au début. Coqueluche datant de 15 jours ; 23 à 24 quintes avec reprise par jour, plus fréquentes la nuit ; ulcération linguale. Traitement commencé le 29 septembre. Dès le 1er octobre, amélioration ; le 3 l'enfant part guérie.

Obs. XII.—Enfant de 5 ans, entré le 12 décembre 1895. Enfant délicat, portant des stigmates de syphilis congénitale et de rachitisme. Coqueluche violente ; 20 quintes par jour ; ulcération linguale, disparition de la coqueluche le 22 décembre. Reste l'adénopathie qui sera traitée, ainsi qu' une tuberculose au début découverte au cours du traitement.

Obs. XIII.—Enfant de 9 mois, présenté le 24 décembre 1895. Coqueluche datant de 15 jours ; une quinzaine de quintes par jour ; guérison en 5 jours.

Obs. XIV.—Enfant de 2 ans : coqueluche datant de 8 jours ; avec quintes déjà suivies de reprise et empêchant le sommeil. Le 5 août 1896 on commence les badigeonnages asaprolés à 1 pour 100 autour de la glotte ; guérison en 8 jours.

Obs. XV.—Nègre de 6 ans. Coqueluche datant d' un mois ; 15 quintes quotidiennes très intenses avec menaces d'asphyxie. Badigeonnages et potion contenant 1 gramme d'asaprol. Le traitement est commencé le 28 janvier ; le 6 février guérison.

Obs. XVI.—Fillette de 4 ans, entrée le 4 février 1896 ; hérédo-syphilis ; coqueluche évidente le 15 ; guérison en 4 jours.

Obs. XVII.—Fillette de 8 ans, entrée le 13 février 1896. Coqueluche ayant débuté depuis un mois. Quintes intenses, avec reprise, vomissements. Badigeonnages institués le 14 au matin ; le 16 il n'y a plus de quintes.

Obs. XVIII.—Enfant de 3 ans, entrée le 25 février 1896. Coqueluche datant de 5 mois, encore violente : 20 quintes par 24 heures. Ulcération linguale, adénopathie trachéo-bronchique. En 8 jours succès complet des badigeonnages asaprolés. Reste l'adénopathie qui sera traitée.

Obs. XIX.—Enfant d'un mois ; signes bien avérées de coqueluche gagnée d'autres enfants qui en sont atteintes. Après 48 heures de traitement les quintes disparaissent.

Obs. XX.—Petite négresse de 3 mois, entrée le 14 avril 1896. Coqueluche datant de 7 jours ; 12 quintes par 24 heures amenant la suffocation. En 3 jours la maladie est enrayée.

Obs. XXI.—Enfant de 2 ans, entré le 27 avril 1896. Coqueluche datant du mois de mars. Quintes au nombre de 25 à 26 par jour, avec vomissements, ulcération linguale. Guérison en une semaine.

Obs. XXII.—Enfant d'un an, entrée le 29 avril. Guérison en une semaine.

Obs. XXIII.—Fillette de 2 ans; coqueluche datant d'un mois; 40 quintes par jour; guérison en une semaine environ.

Obs. XXIV.—Garçon de 26 jours, entré le 2 octobre 1896, malade depuis une semaine; frères atteints de coqueluche depuis trois mois. Le traitement commencé le 8 octobre est suspendu, la coqueluche étant enrayée.

Obs. XXV.—Fillette de 2 ans, entrée le 17 mai 1897; plus de 40 quintes par jour avec menaces d' asphyxie. Ulcération du frein; succès du traitement. Le 26 mai on ne parvenait pas à ramener les quintes en titillant la luette ou en comprimant le thyroïde.

Obs. XXVI.—Fillette de 4 ans, entrée le 4 juillet 1897. Coqueluche datant de 15 jours, enrayée en 5 jours.

Il me seroit aisé d'ajouter ici d'autres observations analogues; celles qui viennent d'être relatées suffisent à mettre hors de doute l'action puissante de l'asaprol contre le germe de la coqueluche dont il enraye le developpement dans les milieux de culture avec autant d'énergie que la résorcine et l'acide citrique. Chez ces 26 sujets d'un mois à 9 ans, la maladie a été guérie ou a avorté lorsque l'application du médicament a pu être faite dès le début, avant la période dite convulsive. Le médicament, doué du reste d'une grande solubilité, a été toujours employé à la dose de 1 pour 100 dans une solution aqueuse. Dans cette proportion, son action locale n'est nullement irritante, et sa saveur légèrement amère devenant promptement douceâtre lui permet d'être très bien toléré par les enfants.

De ce qui précède il ressort donc que l'on possède dans l'asaprol un agent thérapeutique de premier ordre pour combattre la coqueluche.

La solution asaprolée est employée en badigeonnages sur l'arrière gorge et à l'entrée de l'arbre aérien au moyen d'un pinceau à poils fins et à longue hampe courbe. Les attouchements qui, aux prémières séances, réveillent tout naturellement les quintes, finissent par être parfaitement supportés, tout en restant du reste absolument inoffensifs. Ils doivent être pratiqués toutes les deux heures dans la journée. Le procédé opératoire n'a rien de pénible, notamment chez les nourrissons, qui s'y prêtent sans la moindre résistance.

PRIMARY SIMPLE ACUTE ENDOCARDITIS.

A CLINICAL OBSERVATION.

By HENRY HUN, M.D.,

Professor of Nervous Diseases, Albany Medical College.

ALTHOUGH valvular diseases of the heart have been the object of much study and are as thoroughly understood as is any part of clinical medicine, yet the endocarditis which produces them has been, in its active stage, the object of but comparatively few clinical observations. Endocarditis is usually secondary; and its symptoms are either masked by those of the primary disease, or run such a latent course that they escape detection and have entirely ceased when the symptoms of the valvular defect reveal not the active endocarditis, but rather the injury which it has done. Our knowledge then of the commonly occurring secondary endocarditis is very obscure, while a primary acute simple endocarditis has been so rarely described that not only are its symptoms uncertain, but its very existence has been doubted.

Indeed, judging from the statements made in many of the modern text-books of medicine, there is a widespread scepticism as to the possibility of the occurrence of a primary non-malignant endocarditis. Whittaker, who fully adopts the belief, which seems steadily to be gaining ground, that no distinct line can be drawn between the malignant and non-malignant forms of endocarditis, in his very complete article on this disease in *The Twentieth Century Practice of Medicine* (vol. iv., p. 145), states that "endocarditis is, therefore, never primary; it is always a secondary process." In Osler's *Practice of Medicine*, every sentence in which seems to be a condensed summary of a large amount of clinical and pathological experience, is the statement (p. 699, 3d edition) that "simple endocarditis does not constitute a disease of itself, but is invariably found with some other affection."

These statements (and similar ones can be found in many modern text-books of medicine) would certainly be true were endocarditis produced by toxines, but proof has been accumulating rapidly of late

years that acute endocarditis, both simple and malignant, is caused by the growth of bacteria on the endocardium. Thus, to mention one of many similar investigations, Dessy (*Lo Sperimentali*, lii., 1) in thirty-six cases of endocarditis found bacteria in the tissue of the valves in thirty-four. The other two were cases of marantic thrombosis without inflammatory changes in the valves. It seems quite possible that the endocardium, notwithstanding its sheltered position, may, under exceptional conditions, be the primary, not the secondary, seat of this bacterial growth, although such cases are naturally of extreme rarity. The following case seems to show that this theoretical possibility is also a practical one, and in view of the widespread doubt as to the existence of a primary non-malignant endocarditis it seems wise to publish a case of this disease which was under observation not only during its whole course but also before and after it, which appears to challenge these statements of Whittaker and Osler and which seems in other regards also to be worthy of record.

E. B., female. Aged 13 years. Nothing can be learned definitely about her family history further than that her father suffered from chronic rheumatism. She has lived in an Orphan Asylum during the past four or five years, and nothing noticeable has occurred during that time, except that she had a mild but typical attack of typhoid fever in the summer of 1896, the temperature reaching the normal point on August 30th. During this attack the chest was examined and no cardiac lesion was found.

On the night of November 20, 1896, the patient was attacked by a severe pain in the region of the apex of the heart. This pain was very severe and many applications were made without giving her any relief, so that the child could not sleep and passed a very nervous and restless night and was slightly delirious. On the afternoon of the next day, when I saw her for the first time, she was coughing, complaining of intense pain just below and outside the apex beat, had a high temperature and was extremely prostrated. Her prostration suggested pneumonia but her cheeks were pale and presented no flush. She had an eruption of herpes labialis. She was somewhat nauseated. On physical examination nothing could be found except a slight prolongation of the first sound of the heart. The heart's action was rapid and slightly irregular. She was given small doses of morphine for the relief of the pain. The subsequent history is shown by the following excerpts from the clinical record and by the accompanying clinical chart.

Nov. 22. The patient remains in about the same condition, complaining of the intense pain, which is somewhat relieved by morphine and by hot applications, and suffering from nausea and cough. The physical signs are the same as they were yesterday except that there are a few moist râles in the left axilla. The temperature and pulse continue elevated, even more so than yesterday, as can be seen by the accompanying chart. She is taking a teaspoonful of whiskey three times a day and one grain of quinine four times a day.

Nov. 23. The patient complains of slightly less pain. Her nausea has ceased. Her prostration continues to be extreme. Her temperature and pulse continue elevated and the heart's action is irregular and tumultuous. The herpes on the lips are well marked. The râles in the left axilla have entirely disappeared, and there is now heard at the apex a distinct cardiac murmur which is so confused that its exact character cannot be made out. There is no enlargement of the cardiac area of dulness and no accentuation of the pulmonic second sound. The lungs seem to be clear throughout. On abdominal examination nothing abnormal is found.

DR. HENRY HUN. TEMPERATURE CHART.

Name E B Case Number. Date November 1899

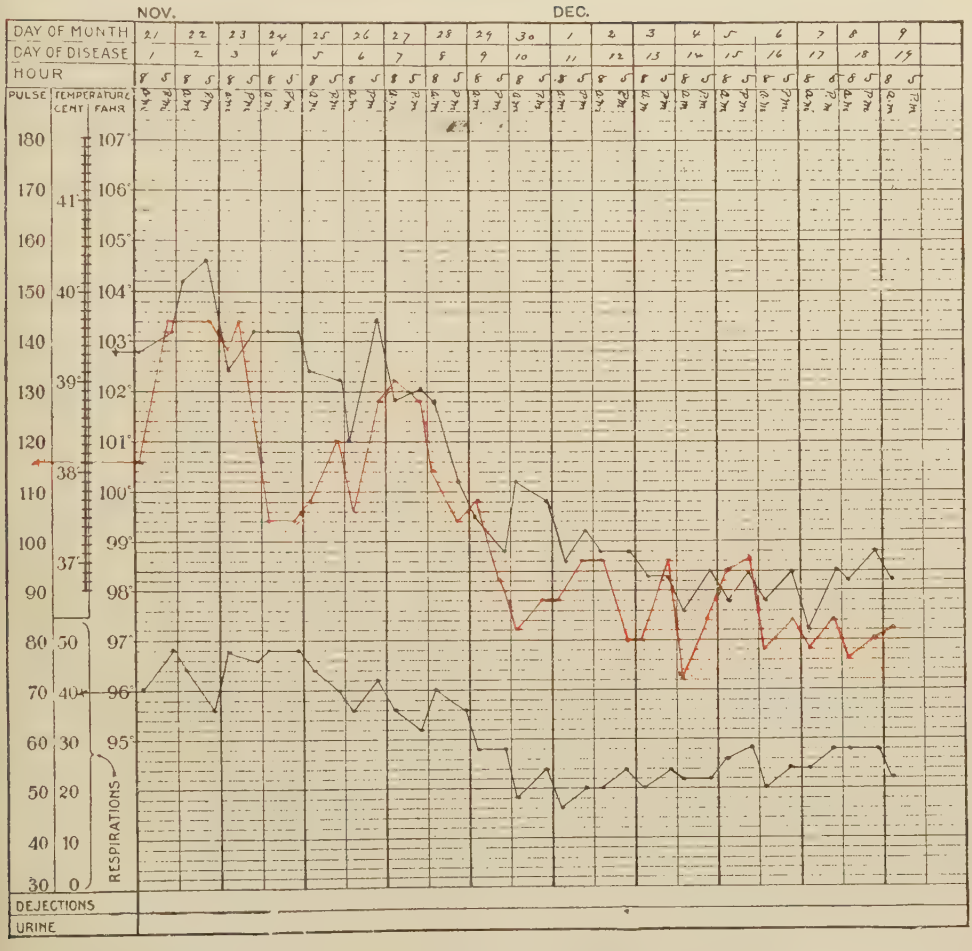


Chart of Temperature and Pulse — Case of Primary Simple Acute Endocarditis.

of six months they could scarcely be felt. During the height of the attack, then, no complications on the part of the lungs, or pleura, or of a rheumatic nature could be detected.

The symptoms were unmistakably those of endocarditis though they exhibited some peculiarities. Thus, the pain present in this case, which was located in the mitral, in contradistinction to the aortic, cardiac area, was unusually severe, and in connection with the high temperature indicated the extreme acuteness of the invasion and may perhaps be characteristic of a primary endocarditis. Certainly acute secondary endocarditis runs its course very frequently without pain. The fever ran a rather irregular course, reached its acme on the evening of the second day, remained continuously high during a week, gradually fell to normal during the next five or six days, and then continued slightly subnormal during the next five days. The pulse was rather unusually high for the temperature, even in a child, during the first three days and was irregular. The prostration, herpes labialis, and course of the temperature, pulse, and respiration were all such as might have occurred in a case of pneumonia.

The results of auscultation were quite interesting. During the first two days nothing abnormal could be detected, except a slight prolongation of the first sound at the apex. On the third day a murmur could be heard at the apex, but so confused that its exact nature could not be determined. On the fifth day this murmur was distinctly systolic and mitral regurgitation seemed to be present, but on the next day this systolic murmur was replaced by a presystolic murmur of stenosis, which continued to be the only one heard during the next few days, and on the eleventh day of the disease the diagnosis of mitral stenosis was confirmed by the existence of an indistinct presystolic thrill, which had each day been sought for and found to be absent. Some three weeks later all confusion had disappeared, and there was evidence of a well marked mitral stenosis and insufficiency with secondary cardiac dilatation and hypertrophy and increased resistance in the pulmonary circulation,—a condition which continued practically unchanged throughout the following year, during which time the child remained in the Asylum under observation.

The symptoms of this case were, then: prostration, fever (with its secondary nervousness, restlessness, and delirium), irregular and rapid pulse and respiration, and severe pain in the neighborhood of the apex of the heart. The valvular lesion caused by the endocarditis gave rise to its characteristic murmurs and subsequently to an increased area of cardiac dulness and an accentuation of the pulmonic second sound.

But it is in its etiology and in the sequence of its lesions that the

chief interest of this case lies. The initial high temperature and all the other symptoms point to an acute infection of the body, doubtless of bacterial origin, and perhaps a late manifestation of some former disease. In regard to this latter possibility, although the child was under careful observation, no illness except that of typhoid fever had occurred during a number of years. Typhoid fever rarely leads to endocarditis even in its active stage; so that there is no reasonable probability that it could be the cause, nearly three months after its subsidence, of an isolated, non-septic endocarditis associated with pain, high fever, and herpes labialis,—the last symptom especially indicating an infection from without the body.

In regard to the nature of any possible infection from without, the family history and the presence of nodules on the fingers subsequent to the attack indicate that the infection may perhaps have been of a rheumatic nature; while the well marked herpes labialis, the general prostration, and the moist râles in the left axilla indicate that it may have been of a pneumonic nature. As between these possibilities the weight of evidence rather favors the belief in the rheumatic origin of this case. But this question, interesting as it is, is of much less interest than the one as to whether the endocarditis was primary or secondary, and on this point, I think, the case gives no uncertain answer, for, whether the nature of the infection was pneumonic or rheumatic, it is clear that the germ did not produce its usual local lesions whether in the lungs or in the articulations and, perhaps in consequence, acted with unusual force on the endocardium and produced much more pronounced symptoms of endocarditis than are usually observed.

Such an occurrence is not at all improbable. It is now well known that typhoid bacilli occasionally pass through the intestinal walls without causing any of the characteristic lesions and produce local manifestations elsewhere and even general septicæmia. In an analogous manner, the germs of pneumonia might pass through the lungs in exceptional cases without producing any characteristic lesion and yet cause a lesion of a vulnerable endocardium in the left side of the heart.

In ordinary articular rheumatism the micro-organism, which is supposed to be its cause, after its entrance into the body, has a long route to traverse before it becomes localized, in consequence of slight traumatism or greater vulnerability, in some special joint or joints. It seems quite possible that this micro-organism might be arrested in its course through the body and become primarily localized upon a cardiac valve rendered vulnerable by an overstrain from work or from too

boisterous exercise. It was the view of our fathers in medicine that rheumatism might in exceptional cases commence in the heart and later manifest itself in the joints. From this view many of the modern text-books of medicine have departed, at least in regard to the acute form, with scarcely adequate cause, it seems to me. In the case here reported careful examination failed to find any involvement of the articulations, there was no rusty sputum or other characteristic sign of a central pneumonia, and the transitory moist râles in the left axilla were not heard at the very outset of the disease and were more likely to be caused by the already disordered cardiac action than to have any causal connection with the latter.

Of course no clinical case can be absolutely conclusive, and an autopsy in this case, had the child died, might have revealed something quite unexpected and shown a primary local infection remote from the heart. As it is, however, from purely clinical considerations we are rather forced to the belief that the infection in this case took place at the time of the illness, and that the germs entered the body from outside and were localized primarily in the endocardium, and that it was a case of primary simple acute endocarditis. And as a clinical case of disease in a child it is gladly offered in honor of him who has been for so many years our guide and our inspiration in clinical pediatrics.

HÔPITAL DE ST. GEORGES À CONSTANTINOPLE.

DR. CAV. G. B. VIOLI.

LE meilleur plaisir que je puisse faire à mon confrère et ami c'est, je le sais, de lui parler d'une institution internationale pour enfants que j'ai fondée à Constantinople en juillet 1895. Avant cette date il n'y avait pas en Turquie d'hôpital pour les enfants. En cas de maladie les enfants qui avaient atteint l'âge de 7 ou de 10 ans pouvaient être admis dans les hôpitaux de la nationalité à laquelle ils appartenaient ; mais avant cet âge ils n'étaient reçus que par exception. Pour les gens du pays il n'y avait pas moyen de faire soigner leurs enfants dans un hôpital, ces établissements n'étant que pour les adultes.

Aidé par la Mission de St. Georges qui a une école à Galata, et qui mettait à ma disposition un local, le personnel de service, avec une retribution pécuniaire j'ai cherché à combler cette lacune, mais il fallait d'argent nécessaire pour transformer ce local en un hôpital avec toutes les annexes nécessaires.

La catastrophe armenienne survenue à ce moment et le krack financier de Constantinople empêchaient de trouver facilement les fonds qu'il fallait pour completer l'œuvre. Cependant avec l'assistance de personnes charitables, j'ai pu, le 1er juillet 1895, ouvrir un petit hôpital dit de St. Georges, pour enfants avec 40 lits ; salles de consultation, d'opération, de désinfection, de gymnastique suedoise, massage et électrothérapie et bain ; à l'hôpital était annexée un service de microscopie et de bacteriologie, ainsi qu'un établissement pour la préparation et conservation du vaccin animal. Dans le jardin un pavillon separé contenant 12 lits était destinés aux diphteritiques.

Afin de trouver l'argent nécessaire pour payer les dettes et entretenir l'hôpital, j'ai fondé une société de protection de l'enfance, dont toutes les personnes charitables pouvaient faire partie en souscrivant la somme minimum de 11½ frs. par an. En outre j'ai donné chaque année sous le patronnage de Dames de la Société de Péra une fête dont le produit à été attribué à l'hôpital. C'est ainsi que j'ai pu encaisser

jusqu'aujourd'hui frs. 77,892. qui ont permis de payer frs. 78,794.77 soit pour l'acquittement d'une partie des dettes contractées, soit pour l'entretien des malades à l'hôpital. Parmi les souscripteurs figurent S. M. T. le Sultan, la Reine d'Italie, l'Empereur d'Allemagne, le Roi de Grèce, feu la Baronne de Hirsch.

Ont été soignés à l'hôpital 7380 enfants souffrant; 5363 de maladies internes; 785 de maladies chirurgicales, de maladies des voies urinaires, et de maladies des yeux; 157 de maladies nerveuses, de paralysies, de malformation du squelette; 377 de la bouche, des oreilles et du nez; 428 de la diphterie et croup; 175 de maladies de la peau; 95 de maladies des dents.

Parmi ces malades 807 ont été reçus internes à l'hôpital et y ont passé 13,540 journées.

En outre 3255 enfants ont été vaccinés et revaccinés.

Tous ces enfants ont été soignés gratuitement sans distinction de culte ou de nationalité.

Sur les enfants souffrant de maladies internes 110 avaient la coqueluche. Ils ont été soignés avec des injections de serum de genisse [1] (inoculée soit hypodermiquement soit par injections intraveineuses de vaccin animal) à la dose de 4 ou 20 cent. c. par séance, selon l'âge des malades. Les inoculations n'ont donné lieu à aucune complication. Elles ont, en général calmé les accès de toux. Quelquefois elles ont guéris les malades.

Trois nouveaux-nés, entre le 3ième et 8ième jour de la naissance, souffrant de Tetanos, ont succombé. Ils avaient été inoculés avec 9 c. m. c. de serum antitetanique de l'Institut Pasteur, en trois séances.

Deux enfants, âgés de 5 à 9 ans, affectés de Leucocitemie avec megalosplenie, engorgements de ganglions inguinaux et de la nuque sont morts au 6ième et 13ième mois de la maladie.

Une fillette âgée de 9 ans, ayant un engorgement des ganglions susmaxillaires et inguinaux avec tendance à l'hemorraphilie, est morte le 10ième jour à la suite d'hémorragie cérébrale. La maladie, Leucocitemie, avait débuté après l'excision de l'amigdale gauche qui avait été pratiquée dans un hôpital de la ville.

Treize malades d'appendicite, soignés médicalement, ont donné 11 guérisons. Parmi les malades atteints des maladies chirurgicales trois souffrents d'appendicite suppurée perforation, peritonite consecutive, opérés par laparatomie sont morts.

Onze qui souffraient du mal de Pott ont été soignés par la methode

[1] Ce traitement a été institué après les communications qui ont été faite en 1893 au Congrès Médical de Rome, sur le traitement de la coqueluche par la vaccination des enfants malades.

Callat; mais nous n'avons pu obtenir aucune guérison radicale, pas même une amélioration.

Trois enfants microcephaliques et idiots ont été craniotomisés; malgré la réussite chirurgicale nous n'avons constaté après 12 et 18 mois de l'opération, aucun bon résultat.

Deux malades avec sarcome du rein ont été opérés et sont sortis guéris de l'hôpital.

Vingt-sept atteints de peritonite tuberculeuse avec ascite ont été opérés par laparatomie et ont été guéris par première intention. Nous avons pu savoir que 11 sont morts, quelque mois après, de tuberculeuse généralisée.

Un cas de kyste hydatique primitif, de la plèvre gauche, a été traité avec succès par l'intervention chirurgicale.

Trois enfants, entre 15 jours et 2 mois, qui souffraient d'accès de suffocation, ont été guéris par l'extirpation des végétations adenoids.

Deux cas de laryngospasme ont été guéris avec l'intubation; on a laissé la canule en place 2 ou 3 heures par jour pendant 3 ou 4 jours.

Un enfant de 6 mois affecté de laryngite aïgué suffocante a été guéri aussi avec l'intubation. Le tube est resté 15 jours dans le larynx avec de courtes détubations.

Dix mastoidites consecutives à des otites chroniques ont été traitées par l'evidement complet de l'apophix et de la caisse et ont guéri, à l'exception de trois cas sur les enfants de 15, 18 (double mastoidite), et 20 mois. À l'autopsie on à reconnu chez le premier une infiltration tuberculeuse de la scissure sylvienne avec noyaux tuberculeux du côté opposé à l'oreille malade. Chez le troisième on a trouvé des tuberculs de la grosseur d'une misette dans la substance encefalique.

Sur 17 cas d'abcès retropharingiens ouverts par la cavité buccale, 16 guérisons.

Un enfant est mort par syncope pendant l'examen digital de la tumeur.

Dans la diphterie le serum nous à donné toujours de bons résultats à condition que les inoculations fuissent faites à temps et que la dose du serum fût proportioné à l'âge du sujet et à la gravité de la maladie.

Sur 280 enfants atteints de diphterie grave, souvent compliquée de streptococcus, quelquefois de scarlatine et de varioles qui ont été transportés à l'hôpital entre le 3ième et le 6ième jour de la maladie, et dont trois étaient à toute extremité, les inoculations ont été pratiquées avec du serum de l'institut local dirigé par Mr. le Prof. Nicolls. Nous avons obtenu 86 % de guérisons.

Sur 45 diphteritiques qu'on n'a pu inoculer par suite de l'opposition des parents nous avons en 63 % de guérisons.

Sur 48 enfants que nous n'avons pas cru devoir inoculer parceque la diphterie dont ils étaient atteints était légère nous avons en 48 guérisons.

Douze cas de croup soignés seulement avec le serum : 9 ont guéri.

Trente-sept soignés avec serum, trachéotomie et tubage : 28 ont guéri.

Les malades de diphterie légère constatés aussi bacteriologiquement ont été surveillés de prés et dès que l'affection présentait un caractère d'aggravation l'enfant était immediatement inoculé, de manière que les guérisons sans serum ont été obtenues seulement dans les cas qui se présentaient bénins et qui restaient tels pendant le cours de la maladie.

Si un enfant habitant la campagne et ne pouvant par cela être surveillé se présentait avec des symptômes cliniques de diphterie, de croup même de forme très légère, il était toujours par precaution inoculés avec le serum.

Nous avons fait très rarement des inoculations preventives et dans ce cas sur des enfants à la mamelle qui ne pouvaient pas être separés de la mère qui soignait le malade.

Nous avons prescrit toujours dans la diphterie soit un traitement interne général tonique soit un traitement local antiseptique avec des irrigations de la cavité orale ou pénétrations des parties malades, avec divers médicaments.

Les 3255 vaccinations et revaccinations faits avec du vaccin animal de notre établissement, avec une asepsie rigureuse, n'ont donné lieu à aucune inconvenient.

ACHYLIA GASTRICA SIMULATING HYPERCHLORHYDRIA.

By MAX EINHORN, M.D.,

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IT is well known that achylia gastrica presents a great variety of symptoms. I desire to treat, in this paper, of cases of achylia which as regards their subjective symptoms are almost typical of hyperchlorhydria. In the latter disease the disturbances (pains, distress, feeling of fulness, etc.) appearing usually one or two hours after meals, are caused by too great acidity of the gastric contents. At first sight it seems rather strange that we should encounter the same symptoms in cases of achylia gastrica, *i. e.*, where there is practically no gastric juice and hence no acid present. Still this is so, as will be seen from the observations later on.

In my book on *Diseases of the Stomach*[1] I have already mentioned that pyrosis may be met with in achylia gastrica. Martius[2] in his book on *Achylia Gastrica* likewise cites a case of achylia in which the principal symptoms were constipation and a burning sensation appearing in the gastric region soon after meals and lasting from half an hour to one hour. In these cases the burning sensation and other symptoms found in hyperchlorhydria (pains, distress, etc.) are certainly not due to irritation of the gastric mucosa by acid substances, for these are entirely absent. It may be that the coarser particles of food, especially after the escape of fluids from the stomach, which occurs in achylia at an early period after meals, excite the mucous membrane mechanically in an intense manner by rubbing against it, and this may be felt as burning or pain. That the ingestion of water would ease this sensation is quite evident. The added fluid mingles with the solid particles lying in the stomach and thus lessens the mechanical irritation. But why the addition of food (even solid substances) should give relief to the pain and burning in some of these cases

[1] Max Einhorn: *Diseases of the Stomach*, first edltion, 1896, p. 418.

[2] F. Martius: *Achylia Gastrica*, 1897, p. 55.

requires further explanation. It may be that the act of eating, as such, fully occupying the mind, draws the attention away from the stomach and therefore the pain or discomfort is allayed (in reality forgotten).

Cases of achylia gastrica presenting subjective symptoms almost identical with hyperchlorhydria are not very rare. I have observed six cases of this type during the present year. Inasmuch as such cases have not, according to my knowledge, been spoken of in literature, I think it of interest to describe them in detail.

CASE 1. APRIL 4, 1899.

Dr. B. W., 54 years old, has been troubled for many years past with dyspeptic symptoms. During the last two years his condition has grown considerably worse. Patient complains of a feeling of tightness and in conjunction with it of slight pains in the gastric region, appearing one and a half to two hours after meals and lasting from one to two hours. Eructation of gas affords only momentary relief, while the ingestion of food or drink causes the pains to disappear for quite a long while. There is no vomiting. The appetite is good, rather increased; the same can be said of thirst. The bowels are slightly constipated. Patient has lost forty-six pounds in weight within the last year and feels rather weak.

PRESENT CONDITION.

Examination of the chest organs does not reveal anything abnormal. Palpation of the abdomen does not show any great tenderness on pressure. A splashing sound can be produced in the gastric region extending almost to the navel. Knee reflex is present. The urine is free from albumen and sugar.

Examination of the stomach one hour after the usual test breakfast shows: the quantity obtained is quite small (20 cc.); there is very little liquid in the gastric contents, and the particles of roll are coarse and hardly changed.

HCl = 0; reaction neutral; no ferments.

A week later the gastric contents were again analyzed and an identical result found.

CASE 2. APRIL 2, 1899.

Dr. A. S., 42 years old, has been troubled for the last four to five years with great discomfort in his epigastric and gastric regions and a severe burning sensation, appearing one to two hours after meals. Partaking of food always relieves this condition. There is frequent belching, but no vomiting. The appetite is good; the bowels are somewhat constipated. Patient has not lost much in weight but is less strong than formerly.

PRESENT CONDITION.

Lungs and heart do not reveal anything abnormal.

Palpation of the abdomen does not elicit any spot painful to pressure. The splashing sound shows that the stomach extends to three finger's widths below the navel. The urine does not contain albumen or sugar.

Examination of the stomach one hour after Ewald's test breakfast shows: HCl = 0. Acidity = 2. No rennet present.

The quantity of gastric contents obtained was small (25 cc.) and there was very little liquid; the particles of roll were unchanged.

CASE 3. MAY, 1899.

H. B., 33 years old, merchant, had always been well until three years ago, when he had malaria For the last eighteen months he was troubled with pains in the epigastric and

gastric regions, appearing about half an hour after meals and lasting one and a half to two hours. Partaking of food always relieved the distress for a short time. The appetite was good; the bowels were constipated. Patient had lost thirty pounds in weight, and was much weaker than previously.

PRESENT CONDITION.

Patient looks sunburnt but somewhat thin; his lips are slightly pale; tongue rather coated.

The physical examination of the chest organs reveals normal conditions. Examination of the abdominal organs shows that the liver is a trifle enlarged, also the spleen; this organ, however, is not accessible to palpation. A splashing sound can be produced in the gastric region extending to the navel. There is no spot painful to pressure. The urine contains no sugar or albumen. The knee reflex is present. Examination of the stomach one hour after Ewald's test breakfast shows: HCl = 0; neutral reaction; pepsin and rennet ferments absent; the quantity of fluid very small, and the particles of roll almost unchanged.

CASE 4. NOVEMBER 30, 1899.

James E. H., 58 years old, was examined for life insurance in 1893 and a murmur at the apex of the heart was found.

In 1895 patient began to vomit soon after meals. Since that time patient was troubled with his stomach, feeling constant pressure and pain in the region of this organ; the bowels were constipated. He grew tired easily and could not walk without becoming exhausted. Recently patient began to suffer from distress and eructation of gas occurring about half to three quarters of an hour after meals, when walking. Besides he was troubled with pains about two to three hours after meals. These pains disappeared after the ingestion of food. His appetite was good and the bowels were slightly constipated.

PRESENT CONDITION.

The examination of the chest shows the lungs in a healthy condition. The heart sounds are clear and there is no murmur. The pulse and temperature are normal. Palpation of the abdomen reveals very slight tenderness in the epigastric region. The stomach is slightly enlarged; the splashing sound can be easily produced over an area extending from the left border of the ribs to about two finger's widths below the navel.

The urine is free from sugar and albumen. The knee jerk is present.

Examination of the stomach one hour after Ewald's test breakfast made January, April, June, and November, 1899, always gave the same result, namely:

HCl = 0; neutral reaction; no ferments; no mucus; particles of roll hardly changed; very little fluid; no remnants of food from the previous day.

The diagnosis of achylia gastrica having been made, the patient was put on a diet rich in starchy and vegetable foods and was given condurango in conjunction with cascara sagrada.

He was advised to take a glassful of water or to eat a cracker with water when the pains came on. He began to grow stronger and gained twelve pounds in weight during the summer. At the same time all the subjective symptoms improved.

CASE 5. SEPTEMBER, 1899.

Rev. A. B., 47 years old, was troubled since his early childhood with dyspeptic symptoms. For the last four years his condition grew worse. Patient began to suffer from pains in his gastric and epigastric regions. These usually appeared two to three hours after meals and were often accompanied by diarrhœa. The ingestion of food generally relieved the pains. During their presence there was a sensation of fulness in the abdomen as if from accumulated gas, — and this was often followed by diarrhœa.

PRESENT CONDITION.

Patient looks well nourished; his lips have a very healthy red color. Tongue is but slightly coated. The chest organs do not show anything abnormal. The examination of the abdomen reveals that the stomach is not dilated, nor is there any distinct tenderness on pressure.

The urine is free from sugar and albumen. The knee jerk is present.

Examination of the stomach one hour after Ewald's test breakfast reveals:

HCl = 0; acidity = 4; no ferments; particles of roll very coarse; very little fluid present; no remnants of food from previous day.

CASE 6. OCTOBER, 1899.

John R., 39 years old, waiter, had been troubled for the past three years with pains which appeared half an hour after meals, and lasted from one to two hours. During this time patient also felt dizzy. The bowels were inclined to diarrhœa. Appetite and sleep were good. There was never any vomiting. Patient had not lost much in weight.

PRESENT CONDITION.

Patient looks well nourished and presents a healthy color of the skin and mucous membranes. Examination of the chest does not reveal any abnormal conditions. Palpation of the abdomen shows slight tenderness on pressure over the epigastric regions. The stomach is not enlarged; the gastrodiaphane shows the greater curvature about three finger's widths above the navel. The knee jerk is present. The urine is free from sugar and albumen.

Examination of the stomach one hour after Ewald's test breakfast reveals: HCl = 0; neutral reaction; no ferments; the particles of roll coarse; the amount of fluid considerable.

In the fasting condition the stomach was found empty.

EPICRISIS.

Cases 1 and 2 are almost typical of hyperchlorhydria; in both there are pains about two hours after meals, and these disappear after the ingestion of food or drink.

Cases 3 and 4 show the period of pain and burning sensation as early as half an hour after meals (in Case 4 also two to three hours after meals). While this is not the rule for hyperchlorhydria, in many cases of this disease, however, especially if it is very marked, the pains appear just as early.

Case 5 suffers from pains two to three hours after meals which are followed by diarrhœa. The latter symptom is not exactly characteristic of hyperchlorhydria, but is met with occasionally there also.

In Case 6 the pains appear half an hour after meals, last for about two hours and are combined with dizziness. This symptom I have also now and again met with in hyperchlorhydria, although as a rule somewhat later after meals.

In all the six cases described, the distress, burning, or pains promptly disappear after the ingestion of food. This circumstance is the most characteristic feature of hyperchlorhydria, and we find it here also.

The diagnosis in these cases has been made after repeated examinations of the gastric contents, which showed the characteristic features of achylia gastrica.

Several of these patients, particularly the first two, who were physicians, had at first treated themselves for hyperchlorhydria but without deriving any benefit. After the correct diagnosis had been established, they improved very promptly.

The importance of examinations of the gastric contents for diagnostic purposes is evident from the above observations.

The prognosis of this special variety of achylia is not different from that of other forms of achylia gastrica and is good, unless grave complications especially of the intestine develop. As a general rule, improvement is observed. As a matter of fact, all my cases during this year were benefited by appropriate treatment to such a degree that they finally presented almost no subjective symptoms whatever.

Besides the usual treatment of achylia, the cases described require special attention for their hyperchlorhydric symptoms. The use of water (half a glassful or a glassful) about one or two hours after meals, just at the time the distress is experienced, appears to be very rational. The water acts as a diluent and diminishes the rubbing of solid particles against the gastric walls (as already mentioned above). Experience teaches that in many of these cases this simple means brings relief. In a few instances milk and crackers were given between meals also with benefit. If these measures are not sufficient, the bromides will then be found useful, bromide of sodium or strontium being given in twelve-grain doses twice daily.

ON THE DIAGNOSIS OF MALIGNANT ENDOCARDITIS.

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IN accepting the invitation to add a chapter to this Jubilee Volume, it has occurred to the writer that the consideration of a disease not sufficiently studied and not easily recognized might be welcome, and he has therefore determined to dilate on the diagnosis of Malignant Endocarditis.

The physician who has had ample clinical and pathological experience with this condition, will credit the statement that it is frequently overlooked, and that the majority of cases are unsuspected and the diagnosis is made post-mortem.

Of the numerous diseases of the heart, whether primary or secondary, degenerative or inflammatory in character, none are more difficult to recognize, none require more thorough differentiation than do those in which there is deep and usually fatal infection of the endocardium. This statement is true for a variety of reasons. The fact that we are dealing with an *infection* which may be considered *secondary under most circumstances*, and which is not dependent upon any *one* pathogenic organism, makes its recognition difficult. In spite of this statement the study of bacteriology has established the etiology of Malignant Endocarditis and made the recognition of its true nature possible.

The diagnosis is sometimes made difficult because the change in the endocardium follows primary disease without material change of the original symptoms. Such cases present nothing characteristic for several days. Owing to the absence, for a time, of marked physical signs referable to the heart, we are led far from the mark and fail in our diagnosis. The diagnosis is often shaded by the uncertainty of the port of entrance of the mycotic agents. These may enter through an insignificant or serious wound, or the endocarditis may result from an infection benign or malignant. The mildest as well as the gravest primary infections have been noted.

In the cases following the unnoticed and insignificant primary infection, the malignant nature of the endocarditis comes as a surprise. Often the primary disease is unheralded and overlooked. In the endocarditis following the graver infections, one is dazed by the gravity of the primary malady and fails to look for secondary and malignant disturbances. It is true that when these are suspected their positive cause may not be at once recognized.

If we classify, according to the severity of the disease, all forms of endocarditis as either benign or malignant, and add the non-specific malignant form of rheumatic endocarditis described by Litten, we often find the differentiation of the benign and malignant forms of endocarditis, or even the non-septic variety and the malignant, a matter of the greatest difficulty. This is true because the symptoms vary from time to time and the changes are sudden and unexpected. An endocarditis which is at first benign may in the course of a few days end fatally with symptoms of malignant infection, while other cases which have commenced with many most acute and alarming symptoms have proven themselves ultimately to be benign or non-septic, in contradistinction to the malignant and fatal forms of the disease. It is very difficult in practice to draw a line which shall separate the severer benign from the malignant either by clinical or pathological study.

In those cases which begin insidiously, without evidences of primary infection or local symptoms (and they are many), the physical signs appearing late, malignant endocarditis is easily overlooked. Many cases have been seen in which the disease has commenced with positive symptoms of general infection and physical signs of endocarditis, in which the symptoms have varied from time to time sufficiently to give rise to grave doubts as to the benign or malignant character of the complication. The frequency with which the valves of the right heart are involved, particularly the pulmonary artery, and the uncertainty of the recognition of those changes, add further difficulties.

Thus in 209 cases collected by Osler (1) he reports the tricuspid valve involved in 19 cases, the pulmonary in 15, the aortic and mitral together in 41, the aortic alone in 53, the mitral alone in 77, the heart walls in 33, and in 9 cases he found the right heart involved alone. These figures prove the involvement of the right heart out of all proportion to the left-sided disease in the less malignant or other forms of secondary endocarditis.

We may suspect the presence of malignant endocarditis, but to make its diagnosis positive, *ante-mortem, we must have the presence of physical signs referable to the heart, and these we have in the majority of cases before death.* This is the positive statement of Leube (2) to

which we cannot take exception. In spite of all these difficulties, we are justified in concluding that after careful examination and deliberate consideration of all the symptoms, with the aid of bacteriologic investigation and other refinements of diagnosis to which we will refer in the following pages, the diagnosis becomes easier as our experience grows.

It is necessary to bear in mind the facts that we are dealing with secondary infection; that that infection may be due to the presence of one or more pathogenic organisms; that the symptoms are both local and general; that these depend more upon the nature, particularly the malignancy of the infection, than upon the method of its production; that the diseased tissue shows a marked tendency to break down into minute particles which act as the carriers of the infectious material to distant parts, where they give rise to deep changes and numerous symptoms, and that these embolic processes are of the greatest diagnostic importance.

It matters not how few evidences of embolic infarct we have, *if we have any*, and these are associated with limited cardiac changes and the presence of few general symptoms, we must suspect the possibility of malignant endocarditis. The importance of these embolic processes, each a source of infection and producer of symptoms, was demonstrated years ago by Virchow (3) in his original study of embolism, and pathological researches have since accentuated their importance.

To Kirke, who in 1851 called the attention of the profession to the subject under consideration, we owe an everlasting debt of gratitude. Since that time we have learned much of secondary infection of the endocardium and its recognition. Traube (4) in his works before 1860 mentions a case of pyelophlebitis in which the diagnosis of malignant endocarditis was made from the occurrence of occasional chills with secondary infection, and in the same work a second case diagnosticated by Traube is reported by Fraenkel, in which this noted Berlin clinician made the diagnosis of malignant endocarditis associated with recurring chills. Litten (5) and Leyden (6) both dilate very thoroughly on the diagnosis of this disease: the former on the forms of septic endocarditis generally; the latter gives a most exhaustive study of those cases associated with intermittent fever.

Rosenstein (7) showed his familiarity with the subject, particularly the pyæmic forms, at the time of the appearance of Ziemssen's great work. That prince of clinicians, Osler (8), who has done so much to advance our knowledge of internal medicine, has given the profession in his Gulstonian Lectures a masterly review of this subject. The recent article of Dreschfelder (9) in *Allbutt's System of Medicine*, gives our knowledge of the subject at the present time.

Complete as these articles are, they leave the differential diagnosis without sufficient consideration, and the last word has not yet been written on this subject. Indeed, the importance of the subject has never been thoroughly appreciated by the profession. Janeway (10) would have us speak of all forms of endocarditis as endocardial fever, and classifies all forms of malignant disease as (1st) malignant endocardial fever with murmurs; (2d) malignant endocardial fever without murmurs.

If the various clinical types are to be classified according to the character of the associated fever, then the continuous, intermittent, irregular, relapsing, or chronic types appear explanatory. If finally the types are considered sufficiently distinct to justify a division which shall include all symptoms, then the following types deserve to be remembered: I. Intermittent or pyæmic; II. Fulminating intermittent; III. Typhoid; IV. Cerebral; V. Chronic cardiac. This latter classification best represents the clinical types.

I. *Intermittent or Pyæmic Type.*—In these cases there is fever with decided intermission and remission, closely resembling the ordinary malarial intermittent. The importance of this type of the disease has impressed the clinician since Kirke's first paper. Traube (11) dilated fully on the possibility of malignant endocarditis with chills and fever. Leyden (12) in his article, which is the most exhaustive bearing on the intermittent forms of fever, gives an excellent clinical exposition of the subject. Frantzel (13) in his treatise on heart disease attempted to make clear the difficulties met in recognizing these cases. Osler (14) in his Gulstonian Lectures called attention to the frequency of fever of the tertian and quartan type and its resemblance to malarial intermittent. Huebner (15) and Ebstein (16) in recent articles have considered the same conditions and cited cases of great interest.

So many conditions are associated with intermittent fever that it is not surprising if during the early days of the disease, when it follows a mild infection in which there have been few general symptoms with but slight or no evidences of endocardial involvement, we fail to reach an accurate conclusion. Usually there is the preceding history of septic or purulent infection to guide us. This is by no means the rule, for many cases have been seen in which the intermittent fever and heart disturbances gave the first clue to the serious lesion. These latter cases, when of staphylococcus origin, have, besides the intermittent fever, marked leucocytosis, enlarged spleens, often early evidences of pus accumulations, increasing delirium, rapid pulse, moderate albuminuria, occasional hæmaturia and the positive evidence of endocarditis in early changes in quality of the heart sounds, which are, in the

majority of cases, followed by increasing dilatation, insufficiencies, and murmurs.

In these cases there may be early embolic infarcts, cutaneous and deep, or these may be late. The cases of staphylococcus infection have been found to run a more rapid course than do those of gonococcic or pneumonic origin. My experience with streptococcic infection associated with intermittent fever has proved that the physical signs of the disease of the heart are longer postponed than with purulent infection; and the cases are more likely to become chronic. In some of these cases with intermittent fever, where there have been pre-existing heart-lesions and consecutive murmurs, the character of these was often changed, and with this change there were occasional transitory paralyses, sometimes evidences of embolic infarct in the skin, the lung, the brain, kidney, or intestines. Obstructive murmurs are most often changed in character and occasionally become inaudible, for a time at least. Later the physical signs become positive.

A case which proved to be exceedingly interesting from a diagnostic point, was seen a number of years ago in which for several days there had been symptoms of intestinal infection. At the same time the patient had a persistent gonorrhœal discharge; the meatus had been cut and the urethra treated mechanically. Without marked disturbances of any kind, without evidences of constitutional infection, this patient suddenly developed a tertian form of intermittent fever, and ague was suspected. It became evident after the third day of the disease that the prostration was greater than is usual in similar cases of malaria, the delirium was greater, the nervous symptoms were more profound, there was increasing shortness of breath, the heart sounds were rapid and suggestive of dilatation of the left heart with mitral insufficiency. There were enlarged spleen and albuminuria.

After the third chill, in spite of the fact that the tertian type of fever persisted, the temperature was always slightly elevated. Physical signs of auriculo-ventricular insufficiencies with aortic stenosis clinched the diagnosis of malignant endocarditis, after the second week. The right middle cerebral artery became plugged; hemiplegia resulted. There were petechiæ in profusion on the surface. These symptoms persisted. He fell into a typhoid condition, the fever became continuous with occasional chills and sharp rises of temperature, and the patient died in the fourth week. The post-mortem with the bacteriologic examination confirmed the diagnosis. There was mixed infection due to the presence in the blood and on the valves of the gonococcus and streptococcus, the former in the ascendency.

In this case there were no purulent deposits, the spleen was enlarged, and in its centre was found a wedge-shaped infarct. The source of fatal infection was undoubtedly the urethra, possibly the early intestinal indigestion may have been a factor. We failed to find the bacillus coli commune on the valves or in the vegetations. The endocardium of the right heart was everywhere covered with deposit.

A young man, in whom we had recognized mitral obstruction years ago, contracted gonorrhœa. He returned to his home after an absence of several weeks in a miasmatic country, with the ordinary symptoms of intermittent fever. While the gonorrhœa was at its height, he had chills and fever of a quotidian type. The mitral obstruction was recognized

by the physician who treated him while away from home. On his return to Syracuse the fever had abated. He had taken quinine. In a few days there was recurrence of fever. On examination the mitral murmur was no longer audible. This was suspicious. The heart was weak, both ventricles dilated. The patient seemed dazed and prostrated, had considerable pain in the abdomen, a few bloody stools each day, pulse became very rapid, urine contained a small amount of albumin, the spleen was slightly enlarged. The disappearance of the murmur, the marked change in cardiac physical signs, the intermittent fever, the great prostration, the ultimate failure of quinine to control the symptoms, aroused the suspicion of ulcerative change in the endocardium, and the diagnosis of malignant gonorrhœal endocarditis was made.

In the course of a few weeks the chills and fever came at irregular intervals, and soon the body was covered with a petechial eruption. This case finally took on all of the symptoms of the chronic cardiac type, and the patient died after four and one-half months with loud murmurs over all the valves.

Examinations of the blood and vegetations on the valves, bacteriologically, confirmed the conclusion that the gonorrhœal infection had caused the ulcerative changes which were found. The spleen, liver, lung, and kidney showed hemorrhagic infarcts and parenchymatous changes. In this case the disappearance of the obstructive murmur was of great diagnostic import. The cause was easily divined ante-mortem.

Most writers mention the fact that the temperature between the exacerbations in the intermittent type returns to normal. This has not been experienced always. In most cases there is a decided fall of temperature and an approach to the normal, but as in the cases above mentioned, after the constitutional symptoms became pronounced, the temperature never touched the health level. In many of these types the character of the fever changes. It may become continuous, the patient remaining in a typhoid condition, in the course of which frequent erratic chills may precede death.

Cases of malignant endocarditis following puerperal phlebitis are not uncommon. In these the pyæmic type of fever predominates. Whenever a patient suffering from puerperal infection shows small or large ecchymotic spots, with increase in the severity of the chills and sharp rise of temperature, or when petechial eruptions are found after it was supposed that the fever was under control, one must think of the possibility of endocardial infection, and in a number of these cases examination will prove that the suspicion was well founded.

After a few days of regular chills it will be noted that the fever rarely touches a point below 100° or 100½° F., leucocytosis is markedly increased, the spleen is often palpable, anæmia and depression increase, and there is marked asthenia. Occasionally there is bloody urine or evidence of pyelitis. The physical signs of endocarditis become more and more manifest. These patients usually die with cerebral symptoms.

With infection and intermittent fever the sudden onset of brain and other nervous symptoms must always arouse a suspicion of advancing infection, and in all these cases the heart must be repeatedly examined. So manifold have the conditions which lead to intermittent fever been found, that it has at times required days of diligent study to discover the source of the symptoms. The walls of the ventricles have sometimes been plastered with granulations and deeply ulcerated in cases which gave no murmurs until within a few days of death.

An occasional transitory paralysis or aphasia, with intermittent fever, must also give rise to a very strong suspicion of embolic detachment and an infected endocardium. The general symptoms will at first impress the diagnostician in the majority of these pyæmic cases, and in the absence of early heart changes will prompt persistence in repeated examinations which ultimately lead to the rational diagnosis.

We judge from Litten's (17) writings that in the majority of cases which he has seen there have been pus foci. While these are comparatively frequent, they have not been found in as great proportion in our cases as in his. We do not conclude that repeated chills always denote the presence of pus. Large pus accumulations were found in two of our cases post-mortem in which during life there were neither chills nor intermittent fever. Ebstein (18) corroborates this statement.

The *chronicity* of many cases of this group is striking. In some, the history covers four and occasionally six months. A woman in whom the diagnosis of chronic malarial fever had been made by several physicians, recently died of hemiplegia after the fifth month, suffering from the chronic intermittent type of infection. This diagnosis was made positive before the patient had been under observation many days. The fever was always preceded by chills. Finally, characteristic periodicity of malaria was absent and the blood failed to show malaria protozoön. Quinine never influenced the course of the disease.

In this case there was a fine hemorrhagic eruption, which some writers (Janeway, 19) have recently described. There were changes in the right side of the heart, both the tricuspid and pulmonary valves were ulcerated and covered with vegetations; and the left heart was also involved. During life there were retinal hemorrhages and frequent conjunctival ecchymoses. One, sometimes two examinations of the blood before quinine is administered will give the positive information so necessary for diagnosis.

In country practice, where there is no trained microscopist, the administration of quinine during several days and a negative result, gradually increasing area of cardiac dulness with weakening of the first sound of

the heart, and rapid pulse, must make us suspicious of endocarditis, probably of the malignant variety.

In the writer's experience no case of malignant endocarditis has presented in which malaria proved to be the cause. Leyden (20) reports a case with intermittent fever in which he attributes the change in the endocardium to the presence of the malarial organism and septic infection. He states, however, that the organism was never discovered, hence the conclusion ought not to be credited.

Herzog (21) reports the case of a woman thirty-three years of age who suffered from intermittent fever, profuse sweats of a quotidian type in which the malaria hæmatozoön was found in the blood. This patient died without physical signs of heart disease. At the autopsy, ulcerative endocarditis involving the aortic and mitral valves was found, the spleen was enlarged, there was chronic nephritis, numerous emboli, and ulceration of the intestines. As there were no cultures made, and as quinine did not control the disease, it may be concluded that there was no malarial infection, though the writer in the consideration of his cases makes the statement that the ulcerative endocarditis was of malarial origin because the protozoön was found before death.

The majority of cases belonging to the intermittent type may be classified as follows:

1st. Pyæmic or septic form.

2d. Erratic intermittent form.

3d. Late cardiac intermittent form.

4th. Intermittent form with pre-existing heart-lesions.

To the first class belong those cases of pyæmia, particularly following puerperal fever, at times occurring during the puerperium, when the patient is up and about but is still carrying a pus focus. In these cases the chills and intermittent fever are due as much to the pyæmia as to the endocarditis. To the second class belong cases in which there are erratic chills, more or less continuous fever at times disturbed by severe chills. The temperature chart appears zigzag, the chills recur, and finally there is a typhoid condition. In differentiating these cases we are occasionally confronted with the possibility of cholecystitis or cholangitis.

The third class includes cases of intermittent fever without positive evidences of the suspected cardiac disease in which the suspicion is finally confirmed before death. The fourth class includes intermittent fever occurring in patients who have pre-existing heart disease upon which is finally grafted a malignant infection. This latter group includes cases which bear the strongest resemblance to malaria.

II. *Fulminating Intermittent Type.* — This type includes a limited

number of cases which run a rapid course, terminating fatally in from ten to seventeen days, occasionally sooner.

A unique case was seen a number of years ago at Borodino, where for four days the patient had been treated for intermittent fever. The chills recurred daily with considerable regularity, the patient grew rapidly weaker, his great and rapid loss of strength became alarming. No hemorrhages were found on the surface, but a marked mitral systolic murmur was discovered. The symptoms aroused suspicion of malignant endocarditis.

At this time he was conscious and volunteered the information that for thirty-six hours a blur had covered his right eye. A large hemorrhagic infarct filled the eye, and in the course of two or three days the sight was lost. The association of chills, mitral murmur, the hemorrhagic infarct in the eye were sufficient to make the diagnosis positive. The patient died before the end of the second week. The source of infection in this case was never discovered.

Two other rapidly fatal cases were found; once after malignant carbuncle in which the disease ended in ten days; in the second case after acute dysentery in which the disease led to death in seventeen days. In both of these cases there was proof of preceding heart disease. A fourth case was found in a child twelve years of age, with violent chorea; the only case in my practice in which chorea was associated with malignant endocarditis. The chills were followed by hyperpyrexia, the temperature on several occasions before death reached 106° F., there were hæmaturia and cutaneous hemorrhages, delirium cordis with tachycardia, indistinct and muffled heart sounds, death in coma between the 13th and 14th days of the disease.

Osler (22) reports the case of a patient who had chorea and who died of malignant endocarditis. The symptoms were positive and the post-mortem proved the correctness of the original diagnosis. Friis (23) and Goodall (24) report cases of chorea associated with ulcerative endocarditis. The complication is very rare.

In the majority of these fulminating cases there was preceding chronic change in the endocardium upon which fresh infection had been planted. Eberth (25) reports the case of a man who had always been healthy, was seized with rigors and high fever followed by unconsciousness. There were symptoms of typhus with meningitis finally. He died on the second day of the disease. The most fulminating cases have followed staphylococcus infection. Streptococcus and pneumococcus infection was long drawn out as a rule. These results accord with the experiences of Weichselbaum (26).

III. *Typhoid Type.*—The disease may begin and end with typhoid symptoms or this condition may precede or follow any of the other types. Most cases belong to this group. Occasionally the patient remains in a typhoid condition with malignant endocarditis for several days or weeks, then with or without pus formation has erratic chills and again falls into the typhoid state. The pure intermittent cases may gradually merge into a typhoid state and die in that condition.

With symptoms of typhoid fever continuing during several weeks, *without chills*, with more or less looseness of the bowels, enlargement of the spleen, increasing anæmia, slight albuminuria, mental torpor, no

paralyses, no marked physical signs referable to the heart, we may be puzzled.

Such a case was transported to Syracuse after eight weeks of symptoms with the diagnosis of typhoid fever. During this time the temperature had been continuously high with but slight morning remission, without changes in the skin or other evidences of embolism. A soft blowing systolic murmur of mitral origin was audible; over the ankles a few fine petechiæ and one or two on the abdomen were found two days after admission. These complications made the diagnosis of malignant endocarditis easy. This patient continued in a typhoid condition until her death, which resulted between four and five months after the onset of the disease. There was no autopsy, unfortunately.

Far more difficult of recognition are the cases in which patients are usually brought to the hospitals in coma with typhoid symptoms and with no reliable history. The symptoms resemble closely advanced typhoid, meningitis, occasionally uræmia. If there is much tympany or slight jaundice with partial effacement of liver dulness one may become suspicious of acute yellow atrophy. In a large proportion of these cases there are neither small nor large hemorrhagic spots. The presence or absence of albuminuria helps little. The Widal test furnishes a valuable link in the chain of evidence.

Repeated blood counts are necessary, as the presence of leucocytosis favors endocarditis and argues against typhoid. The diagnosis of typhoid fever must be surrendered when that disease is supposed to be at its height if the patient has repeated chills. *If the disease was primarily typhoid, we may be sure of secondary infection if chills persist.* The occurrence of chills with typhoid symptoms is suggestive at least of fresh infection and demands an examination of the heart and parts of the body which are often the seat of embolic infarct. In a few of these cases paralyses suddenly arising with physical signs of disturbed circulation point to malignant endocarditis. These paralyses may be either permanent or transitory.

In central New York we rarely see cases of typhoid fever with well marked initial chills, and their occurrence during the disease is rare. The association of typhoid fever and malignant endocarditis has been so rarely found that chills in the course of typhoid fever are not suggestive of this complication. The anæmia in cases of the typhoid type is much greater than is found during the active stage of typhoid fever.

In malignant endocarditis there is less morning remission of the temperature than is customary in typhoid fever. The pulse is more rapid, softer, has less character, is more likely to intermit, and becomes irregular and arhythmic in malignant endocarditis. The presence of retinal hemorrhages to which Litten (27) called attention is also to be considered. The prodromal period of typhoid is longer and more

characteristic. In the majority of cases the diazo-reaction strengthens the diagnosis of typhoid, but is not considered conclusive.

In suspected cases of the typhoid type repeated examinations for days were rewarded with success. In no case has the patient died without final evidence in physical signs of heart disease. It has occasionally happened that a pulmonary infarct has cleared the diagnosis. Here the sputum became tenacious and bloody with characteristic odor, delirium increased, and evidences of œdema of the lung followed. In two cases there was pulmonary gangrene. The majority of cases due to pneumonia or influenza infection have been of the typhoid type.

A puzzling case which might be considered here was seen in which the diagnosis of typhoid fever had been made. The girl was fifteen years of age, had been sick between two and three weeks, had a continuous fever with morning remission. About the tenth day of the disease she had profuse intestinal hemorrhage, more or less distension of the abdomen, slight enlargement of the spleen, a very rapid pulse, grave anæmia with great prostration. She had a copious intestinal hemorrhage the evening I saw her. The heart was rapid, the sounds muffled and indistinct. The suspicion of endocarditis was at once considered. It was found that there was a far-reaching purpuric eruption. The spots varied in size from a pin point to a split pea. The mind was clear, the pupils were dilated, there was no evidence of retinal hemorrhage.

Typhoid fever, malignant purpura, and malignant endocarditis were considered. Widal reaction was negative. The blood count showed the red blood corpuscles reduced one-half, the whites relatively decreased, diazo-reaction negative. In the course of a few days there were hemorrhages from the buccal mucosa. The mind remained clear; the pulse was persistently rapid, the heart sounds less muffled than before without adventitious sounds. The diagnosis of typhoid fever was eliminated, but to differentiate between malignant endocarditis and malignant purpura required two weeks. The patient made a tardy recovery with a normal heart after a long convalescence, which fact convinced us that she had malignant typhoid purpura, which Hoffman (28) has described so well.

Similar cases in which it has been difficult to diagnose between purpura fulminans, the typhus-angiohæmatique of Mathieu and Gomot, are reported by Oliver (29). In these chronic types it has been difficult to diagnose between splenic anæmia, purpura, and malignant endocarditis.

IV. *Cerebral Type.*—Bramwell (30) suggested the consideration of this type in his work on *Diseases of the Heart.* But few of the cases of the pure cerebral type have been seen by the writer. In all the types of the disease there is a strong tendency to coma and other cerebral symptoms as the disease advances. In many cases of the typhoid

type the patients die in coma. The cerebral type is occasionally met in hospital practice, where the symptoms resemble very strongly those of cerebro-spinal fever or meningitis.

In two post-mortems we found malignant endocarditis with cerebral symptoms after pneumonia, where endocarditis was not suspected. The valves were clean, the ventricular walls studded with vegetations, polypi in one case and ulceration in both. In another case after puerperal fever the patient became unconscious, there had been preceding thrombosis of the femoral vein, death followed in twenty-four hours after admission to the hospital. The post-mortem showed both the right and left hearts the seat of vegetations and ulcers. In these cases the temperature remained above 104° F. Secretions were reduced or suppressed, there were evidences of nephritis, the urine contained albumin and blood.

In two cases convulsions preceded death. In one case there was associated purulent pericarditis. In some of these cases the vegetations were so placed as not to give rise to murmurs.

V. *Chronic Cardiac Type.*—This group includes all cases of chronic endocardial disease in which infection has led to mycotic endocarditis. Goodhart (31) has eloquently spoken of the frequency of endocarditis with chronic valvular disease in the following words: "Patients with chronic sclerotic valves are walking mushroom beds, in common times without spawn, but in periods of epidemics germs enter by various channels, which fertilize in these cases into ulcerative endocarditis; in others, to suppurative processes." Goodhart's paper shows that 61 of 69 cases presented old thickening of the valves, and mention is made of the occurrence of malignant endocarditis with aneurismal disease.

Osler (32) mentions the frequency with which sclerotic disease of the aorta and valves is associated with the disease under consideration. Sir James Paget (33) refers to the frequency with which changed valves suffer from acute disease. In the report of St. Bartholomew's Hospital, including 84 cases of infectious endocarditis seen in that institution from January, 1890, to March, 1897, 51 were found in males, or 60.71 per cent., and 33 in females, or 39.29 per cent. The greatest number were found from the thirtieth to fortieth years of age; most of the cases between twenty and forty. In these cases all but 10 were found either in patients suffering from old cardiac disease or there was positive evidence of an infecting lesion. The tendency of old diseased valves in pneumonics to take on chronic malignant inflammation is surprising.

The experiments made by Wyssokovitch (34) explain, in a measure at least, the predilection which germs have for the diseased endocardium. He found that infectious material introduced in the blood-stream attacked by preference the valves and endocardium which had been traumatically robbed of their protecting endothelium. Oliver (35), as well as the observer just quoted, after a series of

painstaking observations, concluded that the healthy endocardium and its included valves resisted bacterial invasion. It might be mentioned in this connection that to Heiberg (36) belongs the credit of having established the fact that in infectious endocarditis the vegetations on the valves were bacterially contaminated.

The clinical histories of the cardiac type vary materially. After marked changes in pre-existing physical signs, the subsequent course may become atypical, decidedly irregular. Any change of physical signs in chronic valvular disease or the sudden disappearance of a murmur with marked constitutional disturbances, including fever, prostration, and rapid pulse, with or without symptoms of embolic infarct, always make the clinician suspicious of malignant endocarditis. Sudden paralyses following with or without surface emboli make the diagnosis positive.

Differential Diagnosis. In 1881, Litten (37) called attention to a variety of endocarditis which he styled "Non-septic Malignant Rheumatic Endocarditis." The cases are often fatal. The early joint symptoms yield to the preparations of salicylic acid. The non-septic endocarditis never leads to suppuration. Litten believes that these cases present distinct features, must be considered separately, and that they are never identical with malignant endocarditis.

In the favorable cases there is the same tendency to contraction and changes in the valves found in the ordinary less virulent forms. The right heart escapes involvement. The infarcts and metastases are never malignant, there is great tendency to pericardial involvement, and recurrences of acute exacerbations in those patients who recover are frequent. There is a noticeable absence of paralyses and symptoms of embolism and purulent thrombosis. Death usually results with cerebral symptoms; the albuminuria is of a more transitory character than in malignant endocarditis; the enlargement of the spleen is less marked,—it may be absent.

The following case of *preceding hæmophilia* deserves mention.

The patient, a boy with hæmophilia, twenty years of age, was taken sick while two other members of the family were bleeding. After he had been greatly reduced by loss of blood without high temperature, he developed malignant endocarditis. Typhoid symptoms were continuous. He died in coma, with pulmonary and cerebral infarcts.

The left internal capsule was occupied by a wedge-shaped clot which had caused right hemiplegia before death. Both the right and left hearts were involved. The valves were ulcerated. Unfortunately, cultures were not obtained. This was the only case seen in which the diagnosis of hæmophilia was positive with associated malignant endocarditis.

A few cases of miliary tuberculosis have been seen in which there

was the typhoid condition, delirium cordis, and scorbutus. The absence of leucocytosis, paralyses, and retinal hemorrhages helped materially. In some of these cases there was no sputum. In all careful search was made for choroidal tuberculosis. The ophthalmoscope proves a valuable adjunct under these circumstances.

Acute yellow atrophy of the liver with petechiæ may possibly be mistaken for malignant endocarditis where the heart sounds are indistinct or murmurs are present. The very rapid prostration, characteristic facies, rapid contraction of liver dulness, the sudden jaundice, absence of paralysis and the characteristic urinary conditions will always make differentiation easy.

The condition of the heart muscle is of the greatest importance in all forms of malignant endocarditis. Degenerative changes are the rule. Toxæmia causes these. In a few cases small pus deposits have been found in the walls of the heart, and there have been infarcts. Stokes's (38) statement that "in the muscle we find the key to the pathology of the heart" must not be forgotten in this disease, neither can we afford to overlook Romberg's (39) work.

Thayer and Lazear (40) make the following interesting statement: "Myocardial changes have been demonstrated in the majority of the cases of acute ulcerative endocarditis of gonorrhœal origin which have come to autopsy, the most satisfactorily studied instance being that of Councilman."

They also mention characteristic myocardial changes, necroses with hemorrhage, leucocytic infiltration, and embolic abscesses. "In Councilman's case the areas of necroses and suppuration were large and gonococci were found microscopically in the foci."

Blood Examination. In all doubtful cases it is absolutely necessary that a thorough microscopic and bacteriologic examination of the blood be made for purposes of diagnosis, while for thorough scientific study of individual cases such examinations are positively demanded. We have frequently mentioned the aid received from blood counts in our cases. The presence of leucocytosis must always remain a strong link in the chain of evidence.

To differentiate typhoid fever and mycotic endocarditis the white corpuscles must be counted. Sittman (41) claims that the specific germ found in the blood gives decided evidence of the port of entry of the germ causing secondary infection. Thus the presence of the pneumococcus would make us suspicious of the lung and the bacillus coli of the intestines as the seat of original disease.

Goldscheider (42) makes the statement that the presence of pneumococci in the blood must always be considered evidence of profound

infection. In pneumonia, if the blood shows pneumococci and there are evidences of endocarditis, it may be considered to be malignant and the prognosis is absolutely bad. Probably no disease shows a richer development of bacteria in the blood than does malignant endocarditis.

Negative results do not always argue against infection. Grawitz (43) makes the statement that repeated negative results of cultures of the blood make the presence of malignant endocarditis unlikely. The highest count of whites in cases of malignant endocarditis has been found to be 44,200, the lowest 10,000. With this leucocytosis there is a decided reduction in the number of red blood corpuscles.

It is safe to follow Cabot's advice never to omit blood cultures in cases of suspected endocarditis, but where the ordinary methods fail, Sittman's (44) method gives the best results. The blood may be taken from the median basilic vein by a sterilized syringe mixed with melted agar which is immediately plated; large quantities of blood are used so that the culture contains at least one-third blood.

In considering this subject the articles of Blumer and Thayer (45), and Thayer and Lazear (46) cannot be ignored. These are classics. The first article contains the report of a case of gonorrhœal endocarditis in which for the first time the authors obtained during life the gonococcus in pure culture from the circulating blood; the second article is equally exhaustive and gives many facts which prove that gonorrhœa is by no means the innocent disease which many suppose it to be. Our growing experiences with severe cases of gonorrhœa and the ultimate development of malignant endocarditis justify the conclusions reached by recent writers, particularly Thayer and Lazear, that the gonococcus must be recognized as a pure pyogenic organism capable of giving rise to grave local and septic complications. In this connection the student needs to consult the articles of Hale White (47) and Siegheim (48).

Since 1890 the condition of the heart has been noticed in severe febrile gonorrhœa by the writer more carefully than ever before, and the frequency of cardiac complications has been surprising. Culture experiments in suspected cases made with the circulating blood will go far towards establishing our knowledge of general infection resulting from the gonococcus and will in doubtful cases aid in clearing the diagnosis.

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CURARE IN THE TREATMENT OF TETANUS.

By Dr. B. SCHARLAU.

CURARE in the treatment of tetanus is not new, but it seems never to have met with general favor. Billroth, in his book of *Allgemeine chirurgische Pathologie und Therapie*, Berlin, 1871, mentions shortly that it has been used here and there, but that it did not fulfil the expectations. The same is true of all the other remedies; even the tetanus serum failed to act in men as it did in animals. Tetanus patients suffer not only from the spasmodic attacks, from the difficulty in breathing, and from the great nervous irritability, but the danger lies much more in the exhaustion from lack of sleep, and from inanition due to the inability to take a sufficient amount of nourishment, if any at all. Chloral hydrate furnishes the means of sleep and relaxes the contracted muscles, but as soon as its effect fades away the muscles again become rigid and the spasmodic attacks reappear. It was, therefore, only natural to look after a remedy which would relax the muscles, even when the patient was awake, thus allowing a more natural rest and the possibility of swallowing. Curare paralyzes the muscles and will work well, if given in such doses as to counteract the reflex irritation of the muscles without paralyzing them. It is, therefore, certainly dangerous to order curare in comparatively large doses quickly in succession, as Hofmann[1] did. The drug is not of uniform strength and must be tested in animals before giving it to men. I have, therefore, always commenced with small doses, which were gradually increased until the physiological effect was visible. I commenced, for instance, with $\frac{1}{12}$ of a grain in a boy, eleven years old, and increased the following doses to $\frac{1}{10}$, $\frac{1}{8}$, $\frac{1}{6}$, until $\frac{1}{4}$ made the opisthotonus disappear, so that the patient was lying on his spine and was able to open his mouth sufficiently to separate the teeth. After four to six to eight hours the muscles commence to show more rigidity, and then another hypodermic injection is given. With every day the

[1] Hofmann administered to a nine-year-old boy one grain of curare three times within one hour.

patient lives, the prognosis becomes better, the tetanus poison gradually losing its strength. If no natural sleep sets in, it is advisable to give chloral hydrate at night. Thus by giving the possibility for nutrition and rest we may hope for a successful termination of the case, especially if the sixth day after the outbreak has passed.

I have treated three cases of trismus and tetanus in this way in the Mt. Sinai Hospital, and all got well. The first one, a young man, twenty-five years old, I attended in Dr. Heineman's service, and, therefore, do not feel justified in publishing it. The other two occurred in the children's department, and were as follows:

Harry Cohen, eleven years old, was admitted to the hospital on August 31, 1895. About four weeks ago he stepped on a piece of wood while barefooted, and a splinter entered the ball of the right foot, which was not withdrawn for six days; two weeks later the boy complained of pain in right shoulder, soon after the entire trunk was held rigid, and on the following day the neck and arms became involved.

On August 29th, marked trismus and tetanus had developed; the attacks came at frequent intervals, unless child was kept perfectly quiet; every attack lasted about five minutes. On admission the above-mentioned symptoms continued: jaws tightly closed, very marked opisthotonus, legs very rigid, abdomen rigid and tense, urination difficult, temperature changing around 100° F. When I saw the patient on September 1st, I ordered hypodermic injections of curare, to commence with gr. $\frac{1}{12}$, which had to be repeated in increased doses, if the former one had no effect, until the effective dose was reached, and this dose had to be repeated as often as its physiological symptoms had faded away. It required gr. $\frac{1}{4}$ pro dosi to relax the muscular tension to such an extent that the rigidity disappeared, and that patient could separate his teeth and drink. To secure a better night's rest chloral hydrate was given in the evenings. The hypodermic injections of curare had to be repeated at intervals of six to eight hours on the average; after six days, however, the effect became more lasting, and only two hypodermics were required in twenty-four hours. From September 12th one hypodermic daily was given until September 20th, when all medical treatment was discontinued. He received instead a warm bath of thirty minutes' duration every morning. A certain amount of muscular stiffness persisted for some time, even after being out of bed, on September 26th; he took, however, nourishment well, and had a natural sleep. Temperature never rose above 101.5°. He was discharged cured on October 5th.

Louis Cohn, seven years old, had measles five years ago, and pneumonia seven months ago. On October 25, 1898, the teacher sent him home from school, because he could not speak distinctly; he could not open his mouth, and the parents say that at the same time they noticed some stiffness in the legs. The boy went around the house despite the rigidity of limbs, until October 30th, when stiffness increased so much that he went to bed; has not vomited, had no convulsions. On October 31st he came to the hospital; general condition and nutrition fair; tongue dry, red, ulcers at lip and on gums; teeth loose; submaxillary glands enlarged; jaws tightly closed. Arms and legs were held rigid, feet in extreme extension with toes held apart; extreme opisthotonus; the muscles relaxed somewhat at times, but when irritated or disturbed in any way he would at once stiffen out. Pupils reacted to light and accommodation. Reflexes, superficial and deep, were exaggerated. Urine was acid, 1032, no albumen, no sugar, but heavy precipitates of urates. Temperature 101.4°, pulse 132, respiration 42.

The treatment was the same as in the first case, only that curarin was given, and that the effective dose was gr. $\frac{1}{80}$. About thirty minutes after the first injection the mouth was

somewhat opened, arms became more lax, but the legs remained as rigid as before. At night gr. v. of chloral hydrate was given, and the hypodermics were continued as often as necessary. The effect was very marked ; on the following day he could, when under the influence of the drug, open his mouth three quarters of an inch, and had a good voluntary motion of his limbs.

Nov. 2d, 8 P.M.—Patient was sleeping with jaws parted about an inch, left leg flexed at knee, and when sole of right foot was tickled, patient quickly flexed right leg at knee.

9.15 P.M.—Child still sleeping, both legs flexed, respiration easy and regular, mouth slightly open.

10.15 P.M.—Child sleeping, but jaws are held closed, masseter contracted, legs extended and somewhat rigid. When awakened he drew head backward, refused to open the mouth, and cried. A hypodermic of gr. $\frac{1}{60}$ curarin was given, and soon after the patient fell asleep with mouth open and jaws one inch apart ; left leg flexed at knee about 60°, right about 20° ; respiration somewhat sighing in character from mucus in throat and nose. He slept quietly during the night, awakening at times and asking for a drink ; he swallows well with the jaws separated about an inch. The course of the disease did not change materially from the given facts, curarin always producing prompt effect.

Nov. 5th.—Has slept from 9.30 P.M. to 3.30 A.M. ; muscles perfectly relaxed.

Nov. 6th.—Slept from 10 P.M. to 5.30 A.M. ; his condition is much improved ; no rigidity of muscles at any time, even when his throat was examined ; mouth almost clean, ulcers on gum healed ; sat up in bed for the first time.

Nov. 7th.—Slept very well ; can protrude tongue, though masseters still feel somewhat hard ; bowels move regularly.

Nov. 10th.—Boy eats and sleeps well, moves with perfect ease, climbs up and down chairs freely. At times he walks with scarcely any stiffness, but when he notices that he is being watched, or when he is asked to walk, his gait at once becomes peculiarly spastic, both legs being held rigid at the knees. The improvement, however, continued and he was discharged cured.

ON THE ETIOLOGY OF HEAD-SHAKING WITH NYSTAGMUS (SPASMUS NUTANS) IN INFANTS.

By JOHN THOMSON, M.D.,

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THE condition treated of in the following paper is a functional co-ordination neurosis of a harmless nature which affects young infants and has a short, well-defined clinical course. It has been described by Henoch, Raudnitz, and other continental writers as Spasmus Nutans, and by Gee, Hadden, Aldrich, Peterson, Dickson, and others under such names as Head-shaking, Head-jerking, Head-nodding, and Head-rotating, Gyro-spasm, Nodding Spasm, etc. We have selected two names for use here, Spasmus Nutans, because it has been so widely employed, and Head-shaking, because it seems to describe what takes place rather better than any one of the other English designations. From the various sources mentioned in the list of references, I have gathered details regarding eighty-eight cases; and the following remarks are founded on these and on thirty-five cases of my own (see Table I).

The clinical features have been so carefully given by previous writers that it will not be necessary to dwell much upon them. It may, however, be mentioned that the two cardinal symptoms — involuntary head movements and ocular nystagmus — are both present in the great majority of cases. In some, however, only head-shaking is observed. We also occasionally find nystagmus occurring alone under such circumstances and with such characters as to make it extremely probable that it is of the same nature as that which occurs with spasmus nutans. This, however, cannot as yet be regarded as proven, and therefore such cases of the kind as I have seen have been placed in a separate table.

The intimate connection between the head movements and those of the eyes is illustrated by two facts. When closure of the unsteady eyes (or eye) takes place naturally in sleep, or is artificially produced

TABLE I—THIRTY-FIVE CASES OF HEAD-SHAKING

Number.	Name.	Sex.	Age, in Months, at Beginning of Symptoms.	Age When First Seen.	Month in Which Symptoms Began.	Month When First Seen.	Approximate Duration.	If Rickety.	Other Facts as to General Health, Injury, etc.	Eyes Affected by Nystagmus.	Direction of Nystagmus.	Usual Direction of Head-shaking.	Other Symptoms Present.
1	Ellen M.	F.	6	6	Nov., '89	Dec., '89		Yes.	Began during severe bronchitis.	Both.	Lat. & rot.	Forward.	
2	John D.	M.	9	9	Jan., '90	Jan., '90	2½ mos.	Yes.	Began during convalescence from measles & broncho-pneumonia.	Left.	Lateral.	Lateral.	
3	Mary N.	F.	6	9	Jan., '90	April, '90	+3 "	No.	Very well.	Left.	Lateral.	Lateral.	
4	Marion S.	F.	5	12	Feb., '90	Sept., '90	+8 "	Yes.	Pale, weakly.	Both.	Lateral.	Forward & lateral.	Internal strabismus.
5	Adam C.	M.		13		March, '91		Yes.	Had congenital syphilis.	None.	None.	Lateral.	
6	William S.	M.	6	6	Dec., '91	Jan., '92	6 "	Yes.	Gums tender.	Both.	Vertical.	Forward & lateral.	Nystagmus of upper lids. Shaking of left arm.
7	James P.	M.	11	13	Dec., '91	Feb., '92	+5 "	Yes.	Gums tender.	None.	None.	Lateral.	Facial irritability.
8	Evelyn B.	F.	8	8	Jan., '93	Feb., '93	+2 "	Yes.		Both.	Lateral.	Forward & lateral.	
9	Alice L.	F.	7	8	Dec., '92	Feb., '93		Yes.	Began one month after measles and broncho-pneumonia. Gums tender.	Both.	Lat. & rot.	Forward & lateral.	Hippus.
10	Elizabeth B.	F.	5	7	Jan., '93	March, '93		Yes.		Both.	Lat. & rot.	Lateral.	Hippus.
11	George D.	M.	11	14	Jan., '93	April, '93	3 "	Yes.		Right.	Lateral.	Forward & lateral.	
12	Isabella D.	F.	3	11	Oct., '93	June, '94		Yes.	Gums tender.	Both.	?	Lateral.	
13	John McM.	M.	6	6	Jan., '95	Jan., '95	2 "	Yes.	Began one month after bronchitis. Gums tender.	Both.	Lateral.	Lateral.	
14	John B.	M.	8	9	Jan., '95	Jan., '95		Yes.	Began one week after a fall.	Both.	Rotatory.	Lateral.	
15	Marion H.	F.	10	13	Jan., '95	April, '95		Yes.	Anæmic. Eczema capitis.	Right.	Rotatory.	Lateral.	
16	James C.	M.	7	13	Dec., '94	May, '95	+6 "	Yes.	Died in a convulsion.	None.	None.	Lateral.	Laryngismus. Convulsions.
17	Ethel B.	F.		8		May, '95		Yes.		Both.	Lateral.	Lateral.	
18	Ellen D.	F.	8	9	Jan., '96	Feb., '96		Yes.	Began during pneumonia.	Both.	Rotatory.	Lateral.	Hippus.
19	Kate McG.	F.	8	11	Dec., '95	March, '96		Yes.	Began soon after a fall.	Right.	Lateral.	Lateral.	Facial irritability.

20	George P.	M.	8	9	Jan., '97	Feb., '97		Yes.	Began 3 days after a fall.	Both.	Lateral.	Lateral.	
21	William V.	M.	5	11	Aug., '96	Feb., '97		Yes.		Both.	Rotatory.	Forward.	Facial irritability. Laryngismus.
22	Ellen H.	F	8	21	Jan., '97	Feb., '97	+13 mos.	Yes.		Both.	Rotatory.	Lateral.	Hippus.
23	Katie B.	F.	4	5	Jan., '97	Feb., '97	+ 4 "	Yes.		Both.	Vertical.	Forward & lateral.	Nystagmus of upper lids.
24	Thomas C.	M.	11	13	Jan., '97	March, '97	+ 3 "	Yes.		Both.	Lateral.	Forward & lateral.	
25	Samuel D.	M.	10	12	Jan., '97	March, '97	6 "	Yes.		Both.	Vertical.	Lateral.	
26	Thomas G.	M.	6	10	Dec., '96	March, '97	11 "	Yes.	A Mongolian imbecile.	Both.	Lateral.	Lateral.	
27	Barbara K.	F.	8	11	Aug., '97	Nov., '97		Yes.		Both.	?	Forward & lateral.	
28	David S.	M.	6	7	Dec., '97	Jan., '98	1½ "	Yes.		Left.	Lateral.	Forward.	Shaking of left arm.
29	Elizabeth W.	F.		10		Jan., '98		No.	Bronchitis.	Both.	Lateral.	Forward & lateral.	
30	George S.	M.	10	11	Jan., '98	Feb., '98		Yes.	Began immediately after a fall. Otorrhœa.	Both.	Lateral.	Lateral.	
31	Victoria P.	F.	9	13	Jan., '98	April, '98	9½ "	Yes.		Left.	Vertical.	Forward & lateral.	
32	Nina Y.	F.	12	13	Jan., '99	Feb., '99		Yes.		Both.	Lateral.	Lateral.	
33	Joanna McL.	F.	16	19	Dec., '98	March, '99		Yes.		Both.	Lateral.	Lateral.	
34	Mary D.	F.	6	9	Dec., '98	April, '99		Yes.	Began shortly after vaccination.	Both.	Vertical.	Forward.	
35	Aggie M.	F.		12		June, '96		Yes.		None.	None.	?	

TABLE II—FOUR CASES OF NYSTAGMUS ONLY.

Number.	Name.	Sex.	Age, in Months, at Beginning of Symptoms.	Age When First Seen.	Month in Which Symptoms Began.	Month When First Seen.	Approximate Duration.	If Rickety.	Other Facts as to General Health, Injury, etc.	Eyes Affected by Nystagmus.	Direction of Nystagmus.	Usual Direction of Head-shaking.	Other Symptoms Present.
1	Annie W.	F.	10	10	Nov., '87	Nov.		Yes.	A Mongolian imbecile.	Left.	Lateral.	None.	
2	Richard D.	M.		18		March, '92		Yes.		Both.	R. vert. L. lat.	"	
3	Janet S.	F.	9	11	Dec., '92	Feb.		Yes.		Both.	L. rot. R. lat.	"	
4	Thomas S.	M.	5	9	Dec., '93	March		Yes.		Both.	Lateral.	"	Twitching of facial muscles.

by the application of wadding and a bandage (Caillé, Raudnitz), the head-shaking ceases. Also when the head is passively steadied by the hand the nystagmus increases, or may appear for the first time.

The child has often a peculiar trick of turning his head to one side and staring fixedly out of the opposite corners of his eyes at things. While doing this he has sometimes a peculiar pre-occupied look. It is important, however, to note that not only is the patient's intellect not seriously affected in spasmus nutans, however long it lasts, but also that, however severe it is in degree, it does not produce the slightest dulness and is not followed by the least sign of exhaustion. The movements seem to be intensified by mental action, as when the child's attention is attracted to any object. They cease during sleep.

The diagnosis of spasmus nutans is generally easy. Eclampsia Nutans or the Salaam Convulsion has been at times mistaken for it, but is quite a different disease, being a form of epileptic seizure associated with serious cerebral defect. What connection the head-shaking of infants has with the similar movements occasionally met with under a great variety of circumstances in adults and older children (*e. g.*, chronic hydrocephalus, idiocy, hysteria, acute feverish conditions, etc.), need not be discussed here. It may, however, be said in passing, that head-nodding and nystagmus (separate or combined) lasting during life is obviously an essentially different condition from the transitory neurosis with which we are at present dealing.

The following case illustrates a rare condition occurring in infancy which may give rise to difficulty in diagnosis:

On December 2, 1899, I saw, through the kindness of my friend Dr. T. J. Thyne, a wasted baby boy, twelve months old, who was reported to be suffering constantly from head-nodding. The child, though weakly, and dyspeptic, was evidently quite intelligent. His mother was in a lunatic asylum. The symptoms dated from May or June and were said not to have changed materially since their commencement.

The movements, although similar in direction, were usually much slower and of greater range than those characteristic of ordinary spasmus nutans. There was sometimes a deliberate shaking of the head from side to side. After this had gone on for a few minutes, there would be a pause—apparently for a rest—and then a simple forward nodding would begin. This nodding was usually accompanied by a corresponding movement of the shoulders, and often the whole trunk was swayed backwards and forwards. The rate of the movements varied considerably from time to time. There was never any nystagmus.

The nodding or shaking was evidently commenced each time intentionally—the child seemed to like doing it. After the movements had gone on for some time he looked tired and a little confused, especially if they had been rapid; then there was a pause. If anything attracted his attention while the head-shaking was going on, the movement stopped at once and only began again when the child's interest began to flag. The movements sometimes took place when he was lying down, and they were said to have been frequently observed during sleep.

The nature of this case was obviously quite different from that of

ordinary spasmus nutans. It was not, like that condition, a purely involuntary affection, but seemed rather a sort of "bad habit." Similar voluntary swaying movements backwards and forwards of the trunk are not very uncommon in young babies who are not well looked after, though the head is not usually implicated. They may be associated with masturbation, but are quite as often, I believe, entirely independent of that habit. The "Head-banging" described by Dr. Gee seems to be another closely related condition.

Etiology. We need not consider here the muscles concerned in the production of the head movement, nor yet the nervous tracts involved. With regard to the latter, however, it may be mentioned that the nervous mechanism, whose function is at fault, is evidently that which includes the semicircular canals in its circuit and which regulates the co-ordination of the movements of the eyes with those of the head. Dr. Alex. Bruce tells me that the nucleus of Deiters (one of the end-nuclei of the vestibular nerve lying at the lateral angle of the fourth ventricle) is in all probability the seat of the disturbance. He infers this from its connections with the oculo-motor nuclei and with the anterior cornua of the cord.

Various facts regarding the children and their surroundings have been regarded by different writers as of etiological importance. We shall refer to some of these and then discuss their relative importance.

Age. More than three-fourths of the published cases (including my own) began between four and twelve months old. This corresponds to the period during which the child is slowly learning to co-ordinate the movements of the eyes with those of the head. At the beginning of it he has great difficulty in moving the two sets of muscles together, and even towards the end he has very little skill in following a moving object with his head and eyes.

Cases have been described beginning as early as one month (Schönberg) and six weeks old (Gee), but no details have been published of any commencing later than the twentieth month. In the case of second attacks, however, the symptoms may still be present in the third year.

Sex. In my series, as in others, the female sex predominates (20 to 15). The proportions in previously reported cases were 37 to 25.

Predisposition to Nervous Disease. There seems little to indicate that the families to which the children belong are specially neurotic. I have, however, met with two instances in which two children in the same family were affected with head-shaking (Cases 6 and 28; 8 and 17). Hadden, also, records one instance of this, and Henoch mentions the occurrence of the disease in twins. Seven of my earlier cases were

kept under observation for periods varying from four to seven years. None of them suffered from any special nervous ailment within that time, except one who had enuresis. In a small proportion of the cases there is a history of convulsions or laryngismus.

Mental Condition. Generally, the intellect is quite normal. The only exception to this I have seen has been in the case of so-called "Mongolian" imbeciles. One of my cases of nystagmus alone, and one of spasmus nutans, were of this type. My friend, Dr. J. Pirie, has had another case of head-shaking in an imbecile of the same kind. At the age at which head-shaking develops, "Mongolian" imbeciles are usually not much behind other children in intellectual acquirements. Even then, however, although often active and lively, they are decidedly backward in co-ordinating muscular movements.

Reflex Irritation (Teething, etc.). No one probably now regards dentition as an essential or important cause of spasmus nutans. Henoch, however, maintains that in some cases reflex irritation connected with teething has some influence in its causation; I have seen cases, such as he describes, where the cutting of a tooth was followed by improvement, and the return of irritation in the gum by a distinct relapse. In one case (23), in which the symptoms began at four months, teething had started a month before. Apart from dentition, obvious sources of reflex irritation are very rare.

Debilitating Illnesses. In many cases the symptoms set in during or soon after some severe illness, such as bronchitis, broncho-pneumonia, or measles.

Defective Light in the Home. This seems to be a most important, if not an essential, element in the causation. In miners' nystagmus—a form of the complaint which resembles that in spasmus nutans in many ways—defective light is probably always a determining cause, along with unnatural straining upward of the eyes, with the body in a cramped position, and a bad atmosphere (Graefe). Raudnitz was the first to draw attention to the occurrence of spasmus nutans in dark houses and during the dark months of the year. He points out that the head-shaking seems to be secondary to the nystagmus, and thinks that the latter is due to a strain of the eyes, probably from the infant constantly attempting to look towards the window.

I have collected the following facts, all of which seem best explained on the supposition that deficient light in the home is a necessary predisposing cause.

1. The homes of about half of my cases were visited, and they were invariably found to be very insufficiently lighted. Usually they occupied the lower stories of high houses in narrow, overshadowed streets.

Of those I was not able to visit, a large number were situated in particularly sunless localities. In every instance the house was in a more or less densely populated part of the town and at some distance from any open space. I have never met with a case in a child who lived in the country. In spite of extensive enquiries, I have never heard of, nor yet seen, a single case of the disease occurring in a well-to-do family. Other forms of nervous disease in infants—such as laryngismus and convulsions—are, of course, quite common among the upper classes.

2. In the very large majority of my cases the symptoms began in either December or January. The details of the thirty cases in which the date of commencement is noted are as follows:

July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
0	2	0	1	0	9	17	1	0	0	0	0

On tabulating the dates of commencement in the forty-six previously published cases, in which this point is noted, we find that in them also more began in December and January than in the other months, although the proportions are not so striking.

July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
0	0	2	4	5	9	12	6	6	1	1	0

These facts are best represented in the form of a chart (see p. 72).

Dr. Alex. Buchan, Secretary of the Scottish Meteorological Society, has kindly furnished me with the following statement of the average number of hours of sunshine in each month of the year in Edinburgh:

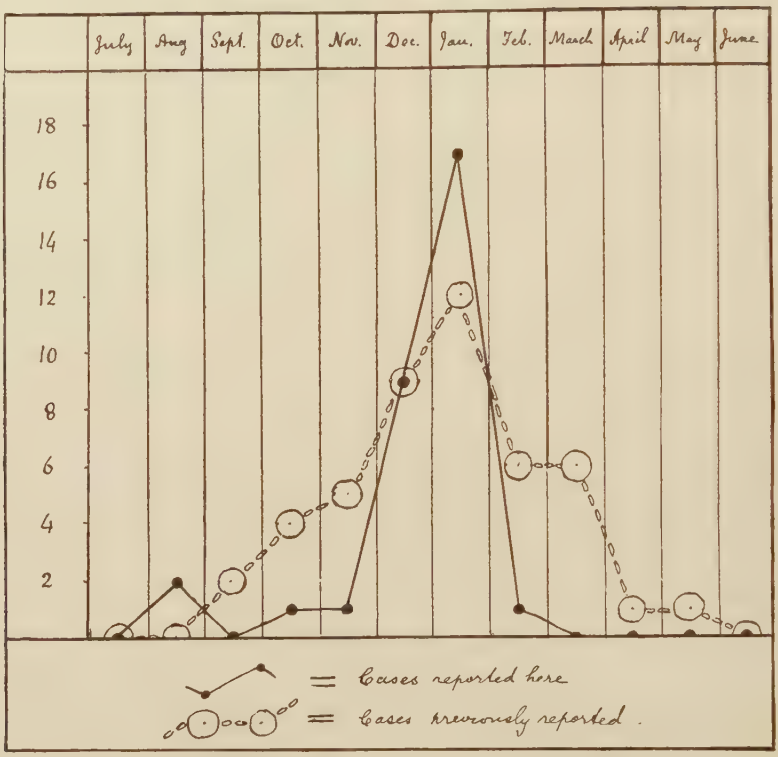
July.	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March.	April.	May.	June.
138	145	104	83	49	24	34	57	98	123	165	157

From this it will be seen that December and January are much the darkest months.

In four cases in which there was a second attack or relapse, after a long period (viz., Hadden's 5th, and Raudnitz's 1st and 9th cases), this took place in every instance in the month of January.

3. We have not enough information with regard to the distribution of cases of spasmus nutans to draw many conclusions. From what little we have, however, it seems tolerably certain, not only that the disease is confined to towns, but also that it is much commoner in some towns than in others. The explanation of this lies probably in the degree of sunlight prevalent, in the amount of it which reaches the

inside of the dwellings of the poor, and especially in the extent to which the children are kept indoors.



The following are some of the facts from which these conclusions are drawn. They have been gathered mostly from previously published papers (especially that of Raudnitz) and from hospital reports.

From the United States only a few cases have been reported in spite of the great interest taken among the profession there in the diseases of infancy. I have not found a single case reported from either France or Italy; and Dr. Pinilla of Madrid tells me that the disease is very rare in Spain. In Vienna it is not at all common (*v. infra*). In Buda-Pesth it is rather more frequently seen (15 cases in 3 years, out of 52,213 patients at the Children's Hospital). In Prague the disease is comparatively common. In Raudnitz's Kinder-Ambulatorium there, there were 8 cases of spasmus nutans in 2 years among 1223 new patients. In Basel, 2 cases occurred among 6154 children. In Berlin, Henoch tells us, he has seen a great many cases. Schönberg published 10 cases seen in 10 years in Christiania. In London, the disease cannot be very rare, considering the number of cases which Hadden saw

within a few years. In Edinburgh, head-shaking is certainly commoner than in many other places. During 11 years (1889-99), among 21,752 children seen by me for the first time at the Sick Children's Hospital and the New Town Dispensary, there were 35 cases of head-shaking, and 3 of nystagmus alone. I have not been able to obtain any statistics about other towns in Scotland, but from opinions expressed by friends in Glasgow, Aberdeen, and Dundee I gather that the disease is not so prevalent in these cities as in Edinburgh.

Head Injury. Instances of head injury having preceded the commencement of the symptoms have been recorded by Hadden, Peterson, Aldrich, and Norrie. In Hadden's 4th case, there were two relapses following falls on the head. In four of my cases there was a distinct history of a fall, in which the head was struck, having occurred shortly before the symptoms began.

Rickets. According to Kassowitz, spasmus nutans is practically always due to rickets, just as laryngismus and the common form of convulsions are. In this opinion he is supported by Schönberg and Hochsinger. Henoch, Hadden, and Raudnitz, on the other hand, record cases in children who were not rickety, although a large proportion of their cases showed signs of this disease.

Out of my 35 cases 33 showed undoubted signs of rickets. The others I was not perfectly sure about. Even if these two were quite free from rickets, 33 out of 35 (94.2 p. c.) is a very large proportion,[1] and it seems impossible to escape the inference that rickets exerts a strongly predisposing influence.

It is questionable, however, whether the connection between rickets and spasmus nutans is of exactly the same kind as that between rickets and laryngismus, convulsions, tetany, and facial irritability (Chvostek's phenomenon). The following are reasons for thinking that it is in some way different:

1. Cases of spasmus nutans, when placed on anti-rachitic regimen, do not (in my experience at least) improve in the strikingly rapid way that is generally observed in the case of these other neuroses.

2. While the four other neuroses are much less frequently found alone than in combination (2, 3, or 4 together), it is comparatively rare to find any of them along with spasmus nutans (4 times in my 35 cases).

3. In some places where rickets and the four associated neuroses are very common, spasmus nutans is rare. The following is one instance of this. Among Kassowitz's out-patients in Vienna the percentage of

[1] Of 3000 children under 2 years examined by me as out-patients between June, 1895, and September, 1899, only 1406 (46.9 p. c.) were undoubtedly rickety.

rickets is greater than in the corresponding class in Edinburgh, and the proportion of laryngismus is large. In six years (1881-86) he saw 370 cases of laryngismus (*Beiträge*, N. F., iv., 1893, S. 63)—an average of 61.6 cases per annum. During 3 years (1896-98) I have seen 61 cases—a yearly average of 20.3. In 8 years (1884-91), however, Kassowitz saw only 9 cases of spasmus nutans (*Beiträge* N.F., i., 1892, S. 165); while, in 11 years 1889-99), there were 35 typical cases among my out-patients.

From a review of these etiological factors we may conclude that the age of the patients, the absence of sufficient light in their surroundings, and the presence of rickets are the most important influences in determining the onset of the disease; likewise that anything which temporarily or permanently lowers the vitality may predispose to its occurrence.

There are many analogies between chorea minor and head-shaking, and we get, I think, an interesting view of the causation of the latter if we group the various etiological factors as Sir. Wm. Gowers does those of chorea.

Chorea, according to Gowers, has three leading factors in its causation. There is, *firstly*, the age. As Sturges used to insist, chorea is a sort of morbidly exaggerated fidgetiness. Naturally, therefore, it almost always occurs in later childhood—the characteristically fidgety age.

Secondly, there is the neurotic element. Under this heading may be included the facts: (*a*) that chorea mostly occurs in neurotic families; (*b*) that there is often a history of over-strain, mental, emotional, or otherwise; and (*c*) that a shock to the nervous system, such as a fright, is often the obvious determining cause of an attack.

Lastly, in a large proportion of the cases there is "a blood-state allied to, but not identical with, that which causes acute rheumatism."

Now, in spasmus nutans we have a similarly complicated causation. There is, *firstly*, a special age (4–12 months). During this the co-ordination of the movements of the eyes with those of the head is growing more perfect, but it is still pre-eminently the age of unsteady eyes and waggling movements of the head.

Secondly, there is the nervous element. Whether it occurs in specially nervous families is uncertain, but it certainly often sets in during the weakness of convalescence and nearly always during the excitable period of teething. The most probable explanation of the striking association of these cases with insufficient light is, as we have seen, that this gives rise to eye-strain and consequent exhaustion of the cerebral

centres. We have also seen that there is sometimes the history of a blow on the head—which means, of course, a fright.

Lastly, the presence of rickets is important as a predisposing cause.

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BEITRAG ZUR LEHRE VOM SCHARLACHFIEBER.

Von A. STEFFEN, Stettin.

MEINE Mittheilungen gründen sich auf über 1200 Fälle, von denen 474 dem hiesigen Kinderspital angehören.

Das Scharlachgift ist uns bis jetzt völlig unbekannt. Es überträgt sich vom Menschen direct auf einen anderen, kann aber auch durch einen dritten, welcher längere Zeit mit Scharlachkranken in Berührung war, oder durch derartige Kleidungsstücke verbreitet werden.

Es wird nicht jeder, welcher die Gelegenheit hat von Scharlach inficirt zu werden, von dieser Krankheit befallen. Es gehört eine besondere Anlage dazu. Es ist nicht selten, dass nur einer oder einzelne in einer Familie an Scharlach erkranken und die übrigen leer ausgehen. Es ist dann vorgekommen, dass letztere sich für immun halten, fremde Scharlachkranke besuchen und von diesen inficirt werden. Es ergiebt sich hieraus, dass das Scharlachgift sich weniger leicht zwischen Körpern mittheilt, deren Gewebe und Zellen von gleicher oder ähnlicher Beschaffenheit sind, als zwischen solchen, welche aus verschiedenen Familien stammen, in welchen sich das Gift zu verschiedener Intensität entwickelt hat. Ich habe dies Vorkommniss nicht blos bei Scharlach, sondern bei verschiedenen Infectionskrankheiten, namentlich bei Pocken beobachtet, wovon ich einen Fall kurz mittheilen will.

Eine junge Frau kommt zum ersten Mal mit einem ganz gesunden Kinde nieder. Drei Tage später erfolgt bei ihr der Ausbruch von Varioloid. Die Amme musste mit dem Kinde in Folge der obwaltenden Verhältnisse in dem Zimmer bleiben, in welchem die kranke Mutter lag. Ich impfte das Kind sogleich reichlich mit guter Lymphe. Die Impfung war ohne Erfolg. Möglicherweise war das Kind durch die mit Pocken inficirte Mutter vor der Geburt immun geworden. Letztere wurde gesund, Amme und Kind blieben gesund. Mehrere Wochen später ging die Amme mit letzterem in das offene Geschäftslokal der Eltern. Hier kam das Kind mit Leuten in Berührung, welche noch

Pockenschorfe im Gesicht und an den Händen trugen. Von diesen wird das Kind inficirt, erkrankt an Variola und stirbt.

Die Anlage an Scharlach zu erkranken kann zu verschiedenen Zeiten des Lebens eine verschiedene sein. Man hat beobachtet, dass Personen, welche als Kinder, trotzdem sie der Ansteckung an Scharlach ausgesetzt waren, frei blieben, auf der Höhe des Lebens und später von dieser Krankheit befallen wurden. Es kann jemand zu verschiedenen Malen und in verschiedener Intensität an Scharlach erkranken. Ich selbst habe dieselben Kinder zwei und drei Male an dieser Krankheit behandelt.

Als Unicum hat mir ein längst verstorbener, durchaus zuverlässiger College berichtet, dass er einen Mann behandelte, welcher jedes Mal, wenn Scharlach sich im Ort epidemisch ausbreitete, von dieser Krankheit heimgesucht wurde. Es war dies wenigstens elf Mal geschehen.

Das Scharlachgift kann bekanntermassen durch Kleidungsstücke, namentlich durch wollene, und zwar oft noch nach längerer Zeit übertragen werden. Ich habe in Bezug hierauf folgenden Fall erlebt, welcher entschieden beweisend ist.

Zwei Schwestern von 12 und 14 Jahren, Töchter eines Landpredigers, befanden sich im Herbst 1887 bei einer alten Verwandten in Pension. Die ältere erkrankte am 7. December an Scarlatina. Ihre Schwester wurde, um sie vor Ansteckung zu behüten, sogleich aus dem Hause in eine bekannte Familie geschicht und blieb von der Krankheit verschont. Die alte Dame, welche die Kranke pflegte, strickte im Krankenzimmer einen wollenen Unterrock. Ich warnte sie, denselben zu verschenken, ehe er nicht gründlich desinficirt sei. Die Kranke genas, beide Schwestern fuhren am Ende des Jahres nach ausserhalb zu ihren Aeltern. Sie kehrten gesund in ihre hiesige Pension zurück. Am 25. Januar, 1888, erkrankte die Jüngere an Scharlach. Die alte Dame hatte ihr den wollenen Unterrock gegeben und klagte sich selbst an denselben nicht haben desinficiren zu lassen.

Es kommen Fälle vor, in welchen nach acht Tagen bis mehreren Wochen die Krankheit Recidiv wird und nach völligem Ablassen des Exanthems, sogar nach beträchtlicher Desquamation des Körpers und fieberfreier Zeit eine neue Eruption von Scharlach auftritt. Es macht den Eindruck, als ob das Gift bei dem ersten Ausbruch sich nicht erschöpft hat und nun sich nochmals geltend macht. Wenn nach ein bis zwei Wochen ein Recidiv erfolgte, habe ich in der Zwischenzeit das Fieber nie vollständig schwinden sehen. Soweit meine Erfahrung reicht, bedingen Scharlachrecidive mit ganz wenigen Ausnahmen eine lethale Prognose.

Ein Recidiv fünf Wochen nach dem ersten Ausbruch des Exanthems habe ich im Jahre 1894 im hiesigen Kinderspital beobachtet.

Ein Knabe von 6½ Jahren wird mit reichlicher Desquamation, verbreiteten Oedemen, Ascites, Transsudat in der rechten Pleurahöhle, beträchtlicher Albuminurie aufgenommen. Allmählig wesentlich und stetig fortschreitende Besserung, Schwinden der Transsudate, doch bleibt die Albuminurie bestehen. Fünf Wochen nach dem Ausbruch des Exanthems findet sich der Rachen und die Tonsillen von Neuem geröthet und geschwellt, Schmerzen beim Schlucken. Zwei Tage später erneuter Ausbruch von Scarlatina, Drüsenschwellung an beiden Seiten des Halses, Oedema facici, beträchtliche Albuminurie, Schwellen von Leber und Milz als Folge einer acuten hochgradigen Dilatatio cordis. Das Recidiv trat mit Fieber bis zu 40.5 auf. Der Knabe wurde gegen meinen Rath nach Hause geholt und ist dort wenige Tage später gestorben.

In der bei weitem grössten Zahl der Fälle stellt der Mund und Rachen die Pforte für die Invasion des Scharlachgiftes dar. Man findet deshalb in der Regel im Beginn, schon vor dem Ausbruch des Exan thems, die Schleimhaut an diesen Stellen geschwellt und mehr oder weniger geröthet.

In nicht seltenen Fällen hat man beobachtet, dass die Aufnahme des Giftes von Wunden aus stattgefunden hat, dass in der nächsten Umgebung derselben das Exanthem erschien und sich von hier über den Körper verbreitete. Ich habe diesen Vorgang namentlich bei Knochenerkrankungen gesehen, wenn bei Periostitis, Coxitis Fisteläffnungen bestanden, zu einer Zeit, als man die heutige Wundbehandlung noch nicht kannte. In einem solchen Fall bei einem 12jährigen Mädchen trat zwei Tage nach Ausbruch des Exanthems unter hochgradigem Fieber der Exitus lethalis ein.

Ich habe ein zartes Mädchen von 1½ Jahren behandelt, bei welcher nach Eröffnung eines Abscesses in der linken Regio inguinalis trotz aller Vorsicht der Scharlachausschlag von dieser Stelle seinen Ausgang nahm und sich dann über den ganzen Körper ausbreitete.

Bei einem Knaben von 7 Jahren, welcher durch Fall sich eine beträchtliche Bisswunde in der Mitte der Zunge zugezogen hatte, entwickelte sich von dieser Stelle Scharlach. Im Verlauf desselben mussten auf beiden Seiten des Halses tiefgehende Abscesse geöffnet werden. Die Ränder der Wunde sowie Heerde in den Tonsillen zerfielen necrotisch. Ausserdem wurde der Fall durch linksseitige Otitis complicirt.

Henoch hat in einem Fall das Exanthem zuerst in beiden Inguinalfalten auftreten sehen, ohne dass eine Wunde vorhanden war.

Ich habe beobachtet, dass bei einem Dienstmädchen, welches von einem an Scharlach heftig erkrankten Kinde inficirt wurde, das Exan-

them zuerst an beiden Händen und Vorderarmen erschien, obwohl diese Körperstellen vorher ganz intact waren.

In einem anderen Fall wurde eine Köchin in den 40ger Jahren von Scharlach befallen, nachdem sie die Leiche eines an dieser Krankheit verstorbenen Kindes gewaschen hatte.

Es scheint, dass in diesen beiden letzten Fällen die Aufnahme des Giftes durch die Haut stattgefunden hat.

Der Ausbruch des Exanthems ist wie die ganze Krankheit sehr vielgestaltig, bald werden einzelne Theile des Körpers, bald der ganze Körper davon eingenommen. Wenn auch in der Regel die Schleimhaut des Mundes und Rachens entzündet gefunden wird, so gibt es doch Fälle, in welchen diese Erscheinung vollständig fehlt, oder kaum angedeutet sein kann. In letzterem Fall pflegt das Exanthem auf der Körperoberfläche ebenfalls weniger entwickelt zu sein. Vor Kurzem habe ich indess eine Ausnahme hiervon bei einem 7jährigen Knaben beobachtet. Es fand sich ein ausgeprägtes, über den ganzen Körper entwickeltes Scharlachexanthem, während Mund und Rachen vollständig frei geblieben waren und geblieben sind.

Die Farbe des Exanthems hält mit der Heftigkeit der Erkrankung gleichen Schritt. In leichten Fällen kann sie ganz blassroth sein, in schwereren mehr oder weniger tief- bis braunroth. Je tiefer die Röthe, um so bedenklicher ist die Prognose und um so mehr ist das Exanthem über den ganzen Körper verbreitet. Bei hochgradiger Entzündung der Haut kann es zu vereinzelten kleinen Blutaustritten in derselben kommen.

Ausnahmsweise kommt es vor, dass die Entzündung des Mundes und Rachens die Diagnose auf Scharlach sichert, während bei aufmerksamster Nachforschung keine Spur von Exanthem auf der Haut nachgewiesen werden kann. Ich habe in einzelnen solchen Fällen Nephritis und Oedeme folgen sehen.

Die Dauer des Exanthems ist verschieden. Sie kann bei schwacher Entwickelung wenige Stunden betragen, in der Regel pflegt es am 4. bis 5. Tage abzulassen. Bei besonders starker Entwickelung habe ich dasselbe noch acht Tage nach dem Ausbruch in voller Blüthe gesehen. Letztere Fälle sind schwer und bedingen eine zweifelhafte Prognose.

Je hochgradiger das Exanthem entwickelt ist, um so früher und intensiver tritt die Desquamation auf und umgekehrt. Ich habe erlebt, dass bei einem jungen Mädchen, welches mit ganz leichtem Ausschlag erkrankt war, erst in der sechsten Woche eine ergiebige Desquamation, und zwar an Händen und Füssen erschien und zu dieser Zeit die Krankheit auf andere übertragen wurde.

Dass es Fälle giebt, in welchen Scharlach mit einem anderen Exanthem zugleich auftritt, ist eine bekannte Thatsache. Ich habe es mit Masern und mit Varicellen vergesellschaftet gesehen. Häufiger kommt es vor, dass Scharlach den Masern oder Varicellen nach 1–2 Wochen folgt oder dass die umgekehrte Reihenfolge stattfindet. Unter den von mir beobachteten Fällen will ich nur folgenden herausgreifen. Bei einem Mädchen von 4 Jahren trat am 2. März, 1896, Scharlach zugleich mit Varicellen auf. Nachdem letztere eingetrocknet waren, erschien vierzehn Tage nach dem Beginn der Erkrankung ein schweres Scharlachrecidiv, welchem das lethale Ende folgte. Die zwei Jahre jüngere Schwester erkrankte am 3. März an Scharlach. Vierzehn Tage später gesellten sich Varicellen hinzu. Das Kind genas nach längerer Krankheit.

Von meinen Beobachtungen über Complicationen des Scharlach will ich nur einige wichtigere zur Sprache bringen.

Es ist bekannt, dass im Verlauf von Scharlach Nephritis mit und ohne secundäre Oedeme und Transsudate in den Körperhöhlen auftreten kann. Nicht in jeder Scharlachepidemie wird diese Complication beobachtet. Es giebt Epidemieen, in welchen fast jeder Fall mit Nephritis verläuft und andere, in welchen diese nur ausnahmsweise zur Beobachtung kommt. Es scheint, dass die Nephritis einer besonderen Beschaffenheit des Scharlachgiftes ihr Entstehen verdankt.

Die Scharlachnephritis zeichnet sich durch diffuse Verbreitung in beiden Nieren aus und kann desshalb bei acutem Auftreten ihre Rückwirkung auf das Herz geltend machen.

Nicht selten nimmt die Nephritis einen haemorrhagischen Charakter an und kann sich unter wechselnden Erscheinungen Wochen und Monate hinziehen. Es gehört zu den Seltenheiten, dass unter diesen Verhältnissen Nierenschrumpfung zu Stande kommt. Nur ein Mal habe ich in einem solchen Fall bei einem Mädchen von $5\frac{3}{4}$ Jahren Nierenschrumpfung mit folgender Hypertrophie und Dilatation des linken Ventrikels nachweisen können.

Das Auftreten von urämischen Erscheinungen bei Scharlachnephritis ist sehr selten. Ich habe im hiesigen Kinderspital folgenden Fall beobachtet: Ein Knabe von 10 Jahren wird mit Scharlach, Nephritis und Oedemen aufgenommen. Anfang der 5. Woche treten urämische Anfälle auf. Ende der 6. Woche findet sich linksseitige Lähmung. Am Abend desselben Tages entwickelte sich Oedema pulmonum, dem ein schneller Exitus lethalis folgte.

Entzündungen der Synovialhäute der Gelenke kommen ziemlich häufig, vereinzelt und multipel im Verlauf von Scharlach vor und werden in der Regel ohne weiteren Nachtheil rückgängig. Bildet sich

Eiterung, so kann Embolie oder Sepsis zu Grunde liegen. In solchen Fällen ist der Ausgang mit seltenen Ausnahmen lethal.

Ich habe im Kinderspital folgenden Fall beobachtet: Ein Mädchen von 12 Jahren wurde im Jahr 1884 im Ablauf von Scharlach aufgenommen und starb nach 14 Tagen. Die Autopsie ergab: Parenchymatöse Nephritis, Bronchopneumonie L. O., beträchtliche Transsudate im Pericardium und Abdomen, und unten in der linken Pleurahöle. Beträchtliche Eiterbildung und Luxation in beiden Hüftgelenken.

Die langen Röhrenknochen erkranken viel seltener in Folge von Scharlach, doch sind dann die angrenzenden Gelenke und Epiphysen meist nicht intact. Neumark berichtet in den Arbeiten aus dem Kaiser und Kaiserin Friedrich Kinderkrankenhause, *Archiv für Kinderheilkunde*, Bd. xxii, über 30 Fälle von acuter infectiöser Osteomyelitis, welche sich in 5 Fällen an Scharlach angeschlossen hatte.

Dass Scharlach mit Erkrankung des Herzens complicirt ist, kann fast als Regel gelten, und zwar betrifft dieser Vorgang in der Mehrzahl das Myocardium, seltener findet man Endocarditis und noch seltener Pericarditis. Man muss eine specifische Einwirkung des Virus auf das Herz annehmen.

In Folge davon kann bei Myocarditis das Herz erschlaffen und Dilatation die Folge davon sein, welche einzelne Abschnitte oder auch das Herz in toto ergreifen kann. Es kann dieser Zustand von dem Gefühl von Herzschwäche mit oft sichtbarem Herzklopfen und einem kaum fühl- und zählbaren Radialpuls begleitet sein. Solche Dilatationen entstehen allmählig, erreichen durchschnittlich nur mässigen Umfang und werden mit dem günstigen Ablauf der Scarlatina in der Regel wieder rückgängig, können aber auch längere Zeit hindurch bestehen bleiben und zu dauernden Störungen der Blutcirculation Veranlassung geben, wenn das Myocardium Veränderungen eingegangen ist, welche nicht mehr rückgängig werden können.

Um vieles bedenklicher erweisen sich die Dilatationen, welche acut im Verlauf von Scharlachnephritis entstehen. Silbermann hat solche Fälle zuerst im *Jahrbuch für Kinderheilkunde*, Bd. xvii, pag. 178, beschrieben. Er hat in drei Fällen zuerst acute Dilatation des linken Ventrikels beobachtet, welcher innerhalb einer Woche beträchtliche excentrische Hypertrophie dieses Herzabschnittes folgte. In zwei Fällen konnte die Autopsie gemacht und die Diagnose bestätigt werden. In zwei anderen Fällen kam es nur zu acuter Dilatation des linken Ventrikels, was in einem derselben durch die Section nachgewiesen werden konnte. Er meint, dass der acuten Dilatation keine excentrische Hypertrophie nachfolgt, wenn das Myocardium durch das Scharlachgift schon zu sehr geschwächt worden sei.

Ich habe bei Scharlachnephritis nur das Zustandekommen von Dilatation, nie von Hypertrophie nachweisen können. Einige bezügliche Fälle habe ich in meinem Buch, *Ueber einige wichtige Krankheiten des kindlichen Alters*, ausführlich beschrieben. In der Regel findet sich nur der linke Ventrikel dilatirt, doch können beide Herzhälften, wenn die Erkrankung des Myocardium den hochgradigeren Process darstellt, von der Dilatation betroffen sein. Das acute Auftreten von Herzdilatation hängt theils von einer Erschlaffung des Myocardium durch das Scharlachgift, theils von der diffusen Entzündung beider Nieren ab, welche dem arteriellen Blutstrom und in zweiter Linie dem linken Ventrikel einen mehr oder minder beträchtlichen Widerstand entgegensetzt. Die plötzliche Steigerung dieser Vorgänge ruft dann die acute Herzdilatation hervor. Unter den Erscheinungen plötzlicher, hochgradiger Dyspnoe kann die Dilatation cordis sinistri in 24–48 Stunden einen solchen Umfang erreichen, dass die Herzspitze im Eingang der linken Axillar gegend steht. In zweiter Reihe kann die rechte Herzhälfte in gleichem Maass ergriffen werden. Dieser Zustand kann schnell zum lethalen Ende führen oder durch rechtzeitige Anwendung von Reizmitteln und kühnen Gaben von Secale rückgängig werden. In letzterem Fall lässt die Dilatatio cordis schnell nach und macht normalen Verhältnissen Platz, auch wenn die rechte Herzhälfte mit ergriffen war. Ich habe die Dilatation in zwei Fällen in 24 Stunden, in drei nach zwei Tagen, in einem nach vier Tagen vollständig schwinden sehen. Man muss aber daran denken, dass dieselbe von Neuem auftreten kann. Ich habe diesen Vorgang nach acht Tagen sich wiederholen sehen.

Nicht selten wird auch das Endocardium vom Scharlachgift ergriffen. Manche Klappenfehler mit ihren Folgen, welche oft erst später entdeckt werden, sind auf diesen Ursprung zurückzuführen. In Folge hiervon kann es zu Embolien kommen, doch sind auch Thrombosen, welche durch die geschwächte Herzthätigkeit und die veränderte Blutmischung bedingt werden, nicht selten.

Als ein Unicum für diese Vorgänge ist die Mittheilung A. E. Pearson und H. Littlewood, welche in dem zweiten Bande von *The Lancet*, 1897, pag. 84, enthalten ist, anzusehen. Dieselbe betrifft einen Knaben von 4 Jahren, welcher am 14. September, 1896, im Spital aufgenommen wird, nachdem nach einigen Vorboten am 10. September die Eruption des Scharlachexanthems begonnen hatte. In beiden Tonsillen fanden sich kleine necrotische Heerde. Am 17. September Blutaustritte in der Haut an beiden Unterschenkeln. In den nächsten Tagen wird die Haut hier livide, hauptsächlich an den Füssen und Fussgelenken, dann erstreckt sich der Livor bis oberhalb beider Kniegelenke. In den Art. femorales konnte keine Pulsation gefühlt werden. Dann trat ein

Sinken der Temperatur in den befallenen Partieen auf und unter lebhaften Schmerzen bildete sich oberhalb beider Kniegelenke eine Demarcationslinie. Gleichzeitig wurde eine mässige Dilatatio cordis constatirt und der erste Ton an der Herzspitze schien etwas rauh zu sein. Am 3. October wurde der rechte, am 10. der linke Oberschenkel amputirt. Am 3. November wurde der Knabe gesund entlassen. Die Untersuchung der amputirten Glieder ergab in beiden gleiche Verhältnisse. Die Aderhäute schienen nicht erkrankt zu sein. Es fanden sich Emboli und Thrombose hauptsächlich in den Arterien und Venen der Kniekehle.

Das Nervensystem kann vom Scharlachgift in hervorragender Weise in Mitleidenschaft gezogen sein. In seltenen Fällen treten mit dem Beginn der Eruption unter heftigem Fieber Convulsionen auf. Die Mehrzahl solcher Fälle verläuft lethal. Ich habe den Tod nach 5–6 Stunden eintreten sehen.

Tetanie im Verlauf von Scharlach gehört zu den sehr seltenen Erscheinungen. Ich habe dies nur ein Mal beobachten können. Es handelte sich um ein sehr zartes Mädchen von anderthalb Jahren, welches an einem schweren Scharlach erkrankt war. Dasselbe wurde Anfangs mit abkühlenden Bädern behandelt. Nach jedem Bade trat ein Anfall von Tetanie auf, welcher mehrere Minuten unter hochgradiger Starre der gestreckten Glieder währte und dann allmählig nachliess und verschwand. Als mit der Anwendung der Bäder aufgehört wurde, erschienen die Anfälle nicht wieder.

Kühn-Uslar hat in der *Berliner klinischer Wochenschrift*, 1899, No. 39, pag. 855, einen Fall von Tetanie beschrieben, in welchem bei einem Knaben von 4½ Jahren im Verlauf von Scharlach fast sechs Wochen hindurch theils allgemeine, theils regionäre tonische Muskelkrämpfe und Muskelstarre mit einer enorm erhöhten allgemeinen Reflexerregbarkeit beobachtet werden konnten. Bei vollem Bewusstsein des Knaben waren etwa 14 Tage hindurch die Erscheinungen von Trismus und Tetanus zugegen. Der ganze Verlauf der Krankheit war fieberlos, Allmählig ist Heilung eingetreten.

Meningitis im weiteren Ablauf von Scharlach scheint ebenfalls sehr selten zu sein. Ich habe nur ein Mal diese Complication gesehen. Das Kind erkrankte in der 6. Woche nach Beginn der Scharlacheruption und ging in wenigen Tagen zu Grunde.

Ein Fall von vorübergehender Lähmung des rechten N. facialis ist mir im hiesigen Kinderspital zu Gesicht gekommen. Derselbe betrifft ein Mädchen von 4½ Jahren, welche am 25. Juli, 1894, mit schwachem Scharlachexanthem und geringem Infiltrat in Mandeln und Uvual aufgenommen war. Am 5. August ist letzteres zum grössten Theil

ausgestossen. Plötzlich wird Nephritis mit vielem Eiweis und Oedema faciei nachgewiesen. Am folgenden Tage ist das Infiltrat im Halse verschwunden, Ascites, beginnendes Oedem der unteren Extremitäten, Lähmung der rechten unteren Facialisäste. Am 8. August auch Parese des oberen Astes des rechten Facialis. In der folgenden Zeit hie und da Arythmie der Herzthätigkeit, der erste Ton vorübergehend unrein. Sensorium dauernd frei. Allmählig nehmen die Transsudate ab und schwinden, Nephritis lässt sich aber noch bis zum 16. September nachweisen. Die Parese des rechten Facialis schwindet allmählig und vollständig. Am 29. September wurde das Kind gesund entlassen.

PACHYMENINGITIS CERVICALIS HYPERTROPHICA? — INTRAMEDULLÄRES, STRANGFÖRMIGES GLIOSARCOM.

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Für die Erkrankungen des Rückenmarkes kommen als anatomische Grundlagen extramedulläre und intramedulläre Laesionen in Betracht, deren differentielle Unterscheidung im klinischen Bilde oft nur sehr schwierig, häufig mit Sicherheit überhaupt nicht möglich ist. Der besseren Uebersicht halber kann man die anatomischen Verhältnisse in folgendes Schema angeordnet den diagnostischen Erwägungen zu Grunde legen.

A. Extramedulläre Laesionen
- *a.* Leptomeningitis
  - acuta
  - chronica
- *b.* Pachymeningitis
  - externa
  - interna haemorrhagica cervicalis
  - hypertrophica
- *c.* Extramedulläre Haemorrhagieen
- *d.* Extramedulläre Tumoren.

Die klinische Differential-Diagnose zwischen diesen Affectionen ist, wie gesagt, mit grossen Schwierigkeiten verknüpft. Nur dem als Pachymeningitis cervicalis hypertrophica (interna) benannten, von Charcot und Soffroy gezeichnetem Krankheitsbilde kommt ein gewisses typisches Gepräge zu, das eine Zeit lang für pathognomonisch angesehen wurde.

Die anatomische Grundlage der von Charcot unter diesem Namen geschilderten Krankheit besteht in der Bildung einer entzündlichen fibrösen Schwiele an der Innenfläche der Dura mater spinalis, welche zur Compression des Rückenmarkes und der austretenden Nervenwurzeln führt. Wieting nennt den Process Meningomyelitis cervicalis hypertrophica, weil es sich dabei meist auch um eine Verwachsung und Verdickung der weichen Häute handelt. Im klinischen Verlaufe der Affection lassen sich zwei Stadien auseinanderhalten. Das erste ist als

die Periode der Schmerzen gekennzeichnet, insofern es durch heftige, continuirliche, in Paroxysmen sich steigernde Schmerzen der Halsgegend und der Körperperipherie gebildet wird. Nun folgt die paralytische Periode, welche durch Muskelatrophieen und trophische Störungen, die von Regenerationsprocessen unterbrochen werden und das Bild einer Querschnittsmyelitis erzeugen können, characterisirt ist. Der Ausgang ist in den meisten Fällen der Tod, doch sind auch Fälle relativer Heilung beobachtet. (Berger, Pietrulla.)

Neuere Beobachtungen haben aber gezeigt, dass zu diesem klinischen Bilde nicht ausschliesslich die anatomische Unterlage durch den pachymeningitischen Process gebildet wird, sondern dass auch Beziehungen zu den Formen der Syringomyelie, welche nicht als reine Hydromyelien, sondern als durch Gefässdegenerationen bedingte Erweichungsprocesse in der grauen und weissen Substanz des Rückenmarks aufgefasst werden müssen, bestehen. Damit deckt also das klinische Krankheitsbild auch einen Theil der im folgenden Schema übersichtlich vereinigten intramedullären Laesionen, welche für die Pathologie des Rückenmarks in Betracht kommen.

B. Intramedulläre Laesionen:
- *a.* Myelitis
- *b.* Intramedulläre Haemorrhagieen
- *c.* Syringomyelie
- *d.* Intramedulläre Tumoren.

Gerade den letzterwähnten beiden Gruppen gehören die Fälle an, die, wie der jüngst von Senator (*Zeitschrift für klinische Medicin*, Bd. xxxv, 1898) publicirte, durch das Vorkommen desgleichen typischen Bildes wie bei der Pachymeningitis cervicalis hypertrophica ausgezeichnet sind. Die Casuistik dieser eigenartigen Affectionen zu erweitern, soll die nachstehende Beobachtung aus dem Kindesalter dienen.

Frida W., 12 Jahre alt, am 11. Nov., 1895, dem Krankenhause zugeführt, aus angeblich gesunder Familie, weder mit Tuberculose, Lues noch Nerven- und Geisteskrankheiten belastet, und bis zum Anfang 1895 vollkommen gesund, gibt an, seit dem Jahre 1894 öfteren Durchnässungen ausgesetzt gewesen zu sein und sich "um kräftiger zu werden" des Oefteren kaltes Wasser über Kopf und Rücken gegossen zu haben. Ende 1894 erkrankte sie unter zunehmender Mattigkeit und schleppendem Gang mit bohrenden Schmerzen im Kreuz und Genick und Steifigkeit des Nackens. Die Schmerzen strahlten über die Schultern und Arme aus das Gehen wurde ihr bald unmöglich, Arme und Beine magerten ab und sie wurde bettlägerig. Allgemeinbefinden nicht gestört. 1895 complete Lähmung des linken, dann auch des rechten Beins und der Arme. Neuralgieen am Hals und Nacken. Im October tritt Incontinentia urinae et alvi auf. Intelligenz während des ganzen Krankheitsverlaufes unbeeinträchtigt.

Status.— Bei der Aufnahme zeigt das Kind eine seinem Alter entsprechende Grösse und einen, abgesehen von den gelähmten und abgemagerten Extremitäten, guten Ernährungszustand. Innere Organe gesund. Schmerzanfälle im Hals und Nacken nach den Armen ausstrahlend. Die auxiliären Respirationsmuskeln in ihrer Function beeinträchtigt, leichte inspiratorische Dyspnoe. Muskulatur der oberen Rumpfhälfte erheblich in ihrer Entwicklung

beeinträchtigt. Fehlen der Schulterwölbung beiderseits durch Atrophie der Deltoides, pectorales und serrati. Schulterblätter flügelartig vom Rumpf abstehend (Serratuslähmung).

Wirbelsäule in der Höhe des 7. Halswirbels kyphotisch. Scoliose nach links. Die Stelle bei Druck nicht schmerzhaft, doch lokalisirt die Kranke dort die *anfallsweise auftretenden Schmerzanfälle*, die auch beim Versuch zu sitzen und den Kopf hochzuhalten auftreten. Kopf kann ohne Unterstützung nicht gerade erhalten werden, fällt vorn über. Rotationsbewegungen frei. Brustwirbelsäule nach rechts scoliotisch, Endenwirbelsäule nach links. Die Rückenstrecker atrophisch.

Obere Extremitäten fast völlig gelähmt, nur mühsam geringe Flexionsbewegungen in den Finger- und Handgelenken auszuführen. Hebung der Arme, Beugung und Streckung im Ellbogengelenk, Pronation und Supination der Hände völlig aufgehoben. Passive Bewegungen schlaff, ohne Widerstand ausführbar. Muskulatur der Ober- und Unterarme stark atrophisch. schlaff. Hände abducirt, im Metacarpophalangealgelenk hyperextendirt, in den Phalangealgelenken flectirt, namentlich links. (Lähmung des medianus und ulnaris; main de prédicateur, en griffe, Klauenhand.) Wölbung der Handrücken abgeflacht, durch Atrophie der musculi interossei zwischen den Metacarpalknochen eingesunken, Daumenballen atrophisch.

Untere Extremitäten bis auf eine geringe Flexion im rechten Knie activ nicht beweglich, schlaff. Muskulatur atrophisch.

Sensibilität im Bereich des Gesichts und Halses nicht gestört. In den oberen Partieen der Brusthaut ausgesprochene Hyperaesthesie. Am übrigen Körper Sensibilität stark herabgesetzt, namentlich die Localisation der Empfindungen aufgehoben. Empfindung von warm und kalt völlig erloschen. In der Bauchhaut, am rechten Unterschenkel und linken Knie vollkommen anaesthetische Bezirke.

Die elektrische Untersuchung ergibt Herabsetzung der Erregbarkeit der Extremitätenmuskeln beiden Stromesarten gegenüber.

Bauchdeckenreflexe erloschen. Unterarmreflexe beiderseits erhöht, Patellarreflexe aufgehoben.

Harn und Stuhl lässt die Kranke unter sich. Sensorium frei, subjectives Befinden bei ruhiger Bettlage auffallend gut.

Diagnose.— Pachymeningitis cervicalis hypertrophica.

Behandlung: Bettruhe, Schmiercur.

Krankheitsverlauf:

12. Dec. Entschiedene Besserung in der Beweglichkeit der oberen Extremitäten, besonders in der Ab- und Adduction der linken Hand. Bewegungen des Kopfes leichter ausführbar. Paraplegie der unteren Extremitäten unverändert.

18. Dec. Fortschreitende Besserung der Bewegungsfähigkeit in den oberen Extremitäten. Pronations- und Supinationsbewegungen in den Unterarmen ausführbar. Leichte Contractionen in den Bauchdecken auslösbar. Flexion des rechten Beines im Hüftgelenk in geringem Masse möglich. Fortbestehen der Paraplegie der unteren Extremitäten und der incontinentia alvi et urinae.

Fortsetzung der Schmiercur:

31. Dec. Bewegungen der oberen Extremitäten erheblich gebessert. Kopfhaltung activ besser. Linker Arm wird im Ellbogengelenk gebeugt und gestreckt, bis zum Kopf erhoben. Hände werden zur Faust geballt. Pat. bemerkt den Abgang von Harn und Stuhl. Paraplegie der unteren Extremitäten unverändert. Patientin erhält Jodkali.

13. März, 1896. Fortbestehende Beweglichkeit der oberen Extremitäten. Besserung in der Paraplegie der unteren. Beine werden in den Hüftgelenken flectirt. Streckung noch unmöglich.

10. April. Pat. kann unterstützt längere Zeit sitzen, wobei Zuckungen in den Beinen auftreten. Patellarreflexe schwach und verzögert auslösbar. Incontinenz wieder stärker.

Anfang Mai: Wiederholung der Schmiercur, worauf spastische Erscheinungen in den

unteren Extremitäten und beiderseits erhöhter Fussclonus beobachtet werden. Elektrische Behandlung.

Mitte Juni: Dembitus am Kreuzbein. Verschlimmerung.

Anfang August: Beweglichkeit in Armen und Beinen mehr und mehr in Abnahme begriffen. Völlige Incontinentia alvi et urinæ. Subjectives Befinden gut.

September: Behandlung mit dem constanten Strom. Nochmalige Schmiercur. Elektrische Erregbarkeit der gelähmten Muskeln für beide Ströme stark herabgesetzt.

3. December: Besserung. Rechter Arm kann im Ellbogen gebeugt und gestreckt, auch pronirt und supinirt und bis zum Kopfe erhoben werden. Im Hand- und Ellenbogengelenk links geringe Bewegungen möglich. Linke Hand noch immer en griffe.

Ende December 1896: Blasenlähmung. Tägliche Katheterisation. Rückgang nach 8 Tagen.

März, 1897: Zunehmende Contracturen in den Hüft- und Kniegelenken. Fortschreitende Atrophie der Muskulatur. Allgemeinbefinden gut, Nahrungsaufnahme reichlich. Fettpolster an den Beinen und Hüften stark entwickelt.

Mai, 1897: Trophische Störungen in der Haut, œdematöse Anschwellungen der Handrücken, starke Schweiss secretion in den Handtellern, wechselnde Erytheme im Gesicht.

September, 1897: Zunehmende Dyspnœ. Hypostasen in den Lungen. Im Anschluss an die dauernde Incontinentia urinæ Cystitis und Pyelonephritis. Zunehmender Kräfteverfall. Steigerung der Dyspnœ. Lungenœdem.

Exitus am 19. September, 1897.

Sectionsbefund:

Katarrhalische Pneumonie in beiden Unterlappen. Eitrige Bronchitis. Lungenœdem. Dilatation des rechten Ventrikels. Geringe Hypertrophie und Dilatation des linken Ventrikels. Septischer Milztumor. Trübe Schwellung der Leber. Parenchymatöse Nephritis. Diphtheritische (nekrotisirende) Cystitis und Pyelitis. Chronische deformirende Arthritis in den Hüft- und Kniegelenken. Hochgradige kyphoskoliose der Wirbelsäule.

Halswirbelsäule lordotisch, Skoliose nach links. Brustwirbelsäule kyphoskoliotisch nach rechts. Lendenwirbelsäule lordotisch und skoliotisch nach links.

Bei Eröffnung des Rückgratkanals von hinten her zeigt derselbe sich im Ganzen erweitert. Die Dura ist *nirgends adhaerent, zeigt an keiner Stelle ihres Verlaufes eine Verdickung oder Schrumpfung.* Dagegen ist im Bereiche der Halswirbelsäule die Pia des Rückenmarks mit der Dura durch leicht trennbare gefässreiche Adhaesionen verwachsen. Die Gefässe der Pia am Halsmark sind vermehrt, geschlängelt und stark injicirt. Ebenso ist die Pia über der Lendenanschwellung des Rückenmarkes reich an zarteren und grösseren, stark geschlängelten Gefässen. Diese beiden gefässreichen Bezirke der Pia stechen durch ihre starke Röthung auffällig gegen die gefässärmere blasse Pia des Brustmarkes ab.

Das Rückenmark selbst (siehe Tafel, Fig. 1 u. 2) erscheint im Vergleich zu einem gleichaltrigen gesunden, im Ganzen etwas verlängert

und voluminöser. Die Volumenzunahme betrifft am Übergang der medulla oblongata ins Halsmark, hauptsächlich den sagittalen Durchmesser, so dass der Querschnitt des oberen Halsmarks die Form eines mit der Spitze nach hinten den Dornfortsätzen zugerichteten Herzens zeigt. Im unteren Hals- und Dorsalmark ist die Volumenvergrösserung mehr durch eine Vergrösserung des transversalen Durchmessers gekennzeichnet, so dass der Rückenmarksquerschnitt querovale Form hat. Im Lendenmark betrifft die Vergrösserung beide Durchmesser, dasselbe erscheint drehrund. Zu diesen allgemeinen Formverhältnissen kommen aber im Hals- und Brustmark sehr erhebliche Abweichungen durch abnorme Verkürzungen des sagittalen Durchmessers, auf welche ich noch zurückkomme. Durch die Krümmungen der Wirbelsäule bedingt haben auch die Krümmungen des Rückenmarks vom Gesunden abweichende Richtung und wesentlich grössere Dimensionen angenommen. So ist das Halsmark stark lordotisch gebogen, während das Brustmark eine stark kyphotische Krümmung angenommen hat, die sich zwischen dem 5. bis 10. Dorsalnerven zu einer scharfwinkligen Abknickung steigert. Von da ab besteht eine leichte lordotische Biegung des Lendenmarks, die die normalen Dimensionen nur wenig überschreitet. Natürlich nimmt das Rückenmark auch in toto an den skoliotischen Verkrümmungen der Wirbelsäule theil.

Nur zum geringeren Theil durch die Verbiegungen der Wirbelsäule, vielmehr in der Hauptsache augenscheinlich durch eine im Innern des Rückenmarks ablaufende Veränderung bedingt, sind die früher erwähnten detaillirteren Veränderungen im Hals- und Brustmark. Zwischen dem 5. und 6., 6. und 7., 7. und 8. Cervicalnerven und zwischen dem 8. Cervical- und 1. Dorsalnerven, ebenso zwischen dem letzteren und dem 2. Dorsalnerven ist das Rückenmark durch besonders am gehärteten Praeparat deutlich hervortretende Querfurchen in eine Reihe den Wirbeln correspondirender Segmente zerlegt. An den Stellen dieser Querfurchen ist der sagittale Durchmesser des Rückenmarks stark verkürzt, sodass besonders zwischen dem 8. Cervicalnerven und 1. Dorsalnerven eine tiefe, quere Einschnürung des Rückenmarks entsteht, die eine nahezu völlige Unterbrechung seiner Masse vermuthen lässt. Ebenso entstehen durch zwei tiefe am Brustmark, etwa in der Höhe des 5. bis 8. Dorsalnerven sitzende quere Furchen, die auch in der Längsrichtung des Rückenmarks ausgedehnter sind, zwei etwa auf die Strecke von je 1 cm. ausgedehnte Bezirke, in denen durch die starke Verkürzung des sagittalen Durchmessers *die Continuität des Rückenmarks beinahe vollständig unterbrochen erscheint.* An diesen Stellen wird der Zusammenhang nur noch durch ein häufiges, an seinen beiden

Rändern wulstig verdicktes Band aufrecht erhalten, dass fast nur aus den verdickten Lamellen der ventralen und dorsalen Piahälfte zu bestehen scheint. Zwischen diesen verdünnten Stellen liegt eine kurze wulstartige Anschwellung von scheinbar erhaltenem Rückenmark. Bis zum letzten Dorsalnerven ist das Brustmark im Ganzen verdickt, dann folgt wieder eine quere Furche, an die sich das verdickte Lendenmark anschliesst, welches in der Höhe seiner grössten Anschwellung durch zwei quere Furchen segmentirt ist.

Die vorderen Nervenwurzeln sind am Halsmark erhalten; sie bilden mit ihren Ursprüngen in der Längsaxe des Markes unregelmässig weit auseinander gerückte Bänder. Am Brustmark sind sie in sehr unregelmässigen Abständen nur theilweise als dünne Fäden erhalten. Die hinteren Wurzeln sind überall als sehr dünne, lang ausgezogne Stränge noch erkennbar. Cauda equina und filum terminale sind ohne Besonderheiten.

Die Consistenz des Rückenmarks ist in seiner ganzen Ausdehnung erhöht.

Auf einem durch das Halsmark gelegten Querschnitt ergibt sich die der ganzen Erkrankung zu Grunde liegende Veränderung des Rückenmarks als eine *strangförmig das ganze Rückenmark durchziehende Neubildung* (s. Tafel, Fig. 3–8). Sie hat im Ganzen die Form eines nach oben bis zur Höhe der unteren Olive reichenden, den Centralcanal daselbst concentrisch umgebenden Stranges, welcher nach seinem oberen Ende zu allmählich sich verjüngt, dagegen im Halsmark eine nach den hinteren Wurzeln zu rasch nach sich vergrössende Ausdehnung erreicht. Im Brustmark erstreckt sich der Tumor unter allmählicher concentrischer Verdickung nach abwärts, wobei in ihm zu den beiden Seiten der Mittellinie eine, wie später noch zu betonen sein wird, vom Centralcanal unabhängige *Höhlenbildung* auftritt. Im Brustmark absteigend und auf das Lendenmark übergehend nimmt der Tumor unter Bevorzugung bald der einen, bald der anderen meist aber der *rechten* Hälfte des Rückenmarksquerschnittes an Volumen zu, mit Ausnahme jener früher erwähnten Stellen, an denen die queren Einschnürungen im makroskopischen Befund beschrieben sind. Dabei nimmt auch die Höhlenbildung unter unregelmässiger Betheiligung der Querschnittshälften zu, sodass *am Uebergang ins Lendenmark ein quergestellter spaltförmiger Hohlraum entsteht*, neben dem sich in der hier vom Tumor besonders eingenommenen rechten Querschnittshälfte mehrere von ihm getrennte Hohlräume ausbilden. In der unteren Lendenmarkhälfte wächst dieser spaltartige Raum zu einer dreiseitig prismatischen grossen Höhle an, die unter allmählicher Verjüngung in der Spitze des conus medullaris ausläuft. Der Inhalt des gesammten

Höhlensystems besteht in einer dickflüssigen gallertigen Masse, die nach dem Lendenmark zu immer mehr bluthaltig wird und zuletzt nur aus Blut besteht.

Das Verhalten des Centralcanals wurde oben bereits gestreift. Im obersten Theile der Geschwulst, am Übergang des Halsmarks in die medulla oblongata hat man auf dem Querschnitt den Eindruck als hätte die Geschwulstmasse sich in concentrischen Schichten um einen central in der Commissur gelegenen spaltartigen Hohlraum entwickelt, den ich bei der makroskopischen Betrachtung als den etwas erweiterten Centralcanal des Rückenmarks ansprach. Mein Assistent, Dr. Seiffert, hat indessen bei Durchmusterung einer grossen Zahl von Schnitten, für deren Anfertigung ich ihm meinen verbindlichsten Dank ausspreche, unzweifelhaft nachgewiesen, dass, wie auch aus den beigefügten Abbildungen hervorgeht, der erwähnte Spalt in Wahrheit *dem Tumor selbst angehört;* welch letzterer sich von der dorsalen Seite des Marks her zwischen die hinteren Wurzeln hereingedrängt und die ventralwärts verschobene Commissur durch seinen Wachsthumsdruck verschmälert und in die Breite verlängert hat. In dieser so veränderten Commissur gelang es Dr. Seiffert bei mikroskopischer Durchsuchung vieler Schnitte in mehreren den gut erhaltenen, an seiner cylindrischen Epithelauskleidung als solchen deutlich erkennbaren *wirklichen Centralcanal aufzufinden.*

Die Structur der Geschwulst selbst, sowie das Verhalten ihrer Gefässversorgung und das Auftreten des Hohlraums in dem Tumor zeigte gleichzeitig in den verschiedenen Höhen erhebliche Differenzen, die sich im Wesentlichen kurz in den folgenden zwei Typen darstellen lassen. Im Halsmark besteht die Geschwulstmasse histologisch aus einem ungemein feinfasrigen, aber auch hie und da stärkere auffällig starre Fasern enthaltendem Netzwerk, in welchem kleine, protoplasmaarme Zellen mit runden sich intensiv färbenden Kernen suspendirt sind. Der histologische Bau entspricht also dem reinen *Neurogliagewebe*, der Tumor muss in diesem Bezirke als ein *Gliom* bezeichnet werden. Dieser Theil des Tumors ist mit reichlichen, aber nicht das physiologische Maass des Glioms überschreitenden Gefässen ausgestattet, deren Wandrungen und Lumen frei von pathologischen Veränderungen sind, höchstens dass nach abwärts im oberen Brustmark eine augenscheinlich auf ein Oedem zurückzuführende Verbreiterung ihrer Adventitia auftritt. Der Spalt ist durch eine einfache Erweichung des Gliomgewebes bedingt, die wohl durch den Druck einer serösen Flüssigkeit von den tieferen Bezirken der Höhlenbildung aus zu erklären ist.

Dem gegenüber zeigt die untere Hälfte des Tumor und zwar gegen

das Lendenmark zunehmend ein wesentlich anderes Bild. Hier wird die Faserung der Grundsubstanz gröber, es überwiegen mehr und mehr die Zellen, die einen grösseren Protoplasmahof bekommen und grössere bläschenförmige Kerne aufweisen. Häufig finden sich die Zellen innerhalb der fasrigen Grundsubstanz zu grösseren Haufen gruppirt. Kurz der Tumor macht den Eindruck einer lebhaften Wucherung seiner zelligen Elemente. In diesen Bezirken entwickelt sich aber ferner ein erheblicher Reichthum an Gefässen, deren pathologische Veränderungen zur Ausbildung des zunehmend sich vergrössernden *Hohlraums* führen. Die sehr zahlreichen Arterien des Tumors sind von breiten hyalinen Scheiden umgeben, welche die Adventitia auf das vier- bis fünffache ihrer normalen Breite vergrössern. Ebenso finden sich um die grösseren Venenlumina Abscheidungen einer hyalinen structurlosen Substanz. Manche Capillaren werden von der sie umgebenden hyalinen Masse förmlich erdrückt. Die meisten dieser Gefässlumina zeigen theils frische rothe, theils wandständige oder auch obturirende weisse *Thromben*. In der Umgebung der verlegten Gefässe und der noch zu erwähnenden Blutungen finden sich reichlich amorphe Klumpen von Haematoidin und mit diesen beladene Rundzellen. Wohl in Folge dieser vielfachen Gefässverlegungen ist es zu einem die Lymphspalten der Geschwulst starkerweiternden Oedem des Gewebes gekommen, das in seiner weiteren Entwicklung dann zu einer *centralen Erweichung* und damit zur Bildung des nach unten an Ausdehnung wachsenden *Hohlraums* geführt hat. Endlich ist die Neubildung an den Stellen ihrer grössten Ausdehnung von reichlichen Stauungsblutungen, die sich auch in den Hohlraum hineinverbreitet haben, durchsetzt. An den bei der makroskopischen Beschreibung erwähnten Einknicklungs- und Segmentirungsstellen ist es dann zu einem Zusammensinken des Rohres und zu einer Annäherung seiner Wandungen gekommen.

Für das Verständniss des klinischen Verlaufes wichtiger sind die Wirkungen der Neubildung auf die nervösen Bestandtheile des Rückenmarks, wie sie durch die Anwendung der Weigert'schen Markscheidenfärbung zur Anschauung gelangen.

Durchgehends zerstört, d. h. der Markscheidenfärbung nicht mehr zugänglich, sind die Hinterstränge des Rückenmarks bis hinauf zu ihren Kernen (s. Tafel, Fig. 3–8). Auf ihrem gesammten Verlaufe ist nirgends mehr leitungsfähige Nervensubstanz nachzuweisen.

Im oberen Halsmark sind auf Querschnitten die Vorderstranggrundbündel und die Pyramidenvorderstrang- und Seitenstrangbahnen comprimirt und verschmälert (Fig. 3). Bei stärkerer Vergrösserung ist eine deutliche Lichtung ihres Faserreichthums erkennbar. Auch vom Gowerschen Bündel und den Kleinhirnseitenstrangbahnen sind

nur noch ganz schmale Reste erhalten, dagegen sind die übrigen Theile des Seitenstranges vollständig marklos, durch die Neubildung substituirt. Die Neubildung kriecht in den noch erhaltenen Faserbündel weiter und zersprengt sie allmählich, so dass in den älteren Theilen des Tumors nur noch unregelmässige varicöse Nervenfaserreste und Markbröckel verstreut liegen.

Die Vorderhörner der grauen Substanz sind stark verkleinert und in ihrer Form verunstaltet. Sie enthalten nur im Halsmark noch spärliche atrophische, fortsatzlose Ganglienzellen mit dunklem sehr geringem Protoplasma. Im unteren Halsmark werden die noch nachweisbaren Ganglienzellen immer spärlicher, im Brustmark werden die Vorderhörner immer schmäler und kürzer, bis sie in der Mitte des Brustmarkes und nach abwärts im Lendenmark kaum mehr eine Andeutung ihrer Form erhalten ist.

Die hinteren Wurzeln sind stark verschmälert und enthalten nur noch ganz spärliche Überreste von markhaltigen Fasern, während in den vorderen Wurzeln noch erhaltene, aber in der Zahl sehr bedeutend verringerte Nervenfasern nachweisbar sind.

Im unteren Halsmark sind neben den Vordersträngen noch spärliche Partieen der seitlichen Grenzschicht und der gemischten Seitenstrangzone erhalten, ausserdem aber auch noch das hintere äussere Feld des Hinterstranges (Fig. 4). Aber auch hier ist überall die Zahl der erhaltenen Nervenfasern eine geringe. An der Stelle der tiefsten Einschnürung sind im Halsmark nur noch Reste der Vorderstränge und auf der linken Querschnittshälfte ein kleiner Theil der äusseren Bezirke der Seitenstränge erhalten.

Weiter nach abwärts tritt nun ein rapider, aber ungleichmässiger Schwund der weissen Substanz ein. Im unteren Brustmark sind nur Theile der Vorderstränge und auf der rechten Seite das hintere äussere Feld des Hinterstranges erhalten (Fig. 5).

Auch am Übergang des Brustmarkes in das Lendenmark ist in der Höhe einer der erwähnten Einschnürungen nur noch ein Theil der Vorderstränge und ein geringer Theil des äusseren Seitenstranges in der linken Querschnittshälfte markhaltig, während die rechte Querschnittshälfte fast gänzlich von der Neubildung occupirt ist (Fig. 6 und 7).

Endlich verliert sich das Mark in der Höhe des zweiten Lumbarnerven fast gänzlich, so dass nur noch unscheinbare Reste der Vorderstranggrundbündel übrig sind (Fig. 8).

Der im Vorstehenden wiedergegebene anatomische Befund dürfte wohl geeignet sein, den klinischen Verlauf mit seinen auffälligen Schwankungen zu erklären. Bei dem langsamen Vorkriechen des

Tumors vom Lendenmark aus nach oben unterliegt es keinen Schwierigkeiten, die zeitweiligen Besserungen im Zustande der Patientin durch den Ersatz der zu Grunde gegangenen Bahnen durch die noch erhaltenen Reste leitungsfähiger Substanz zu erklären, wozu noch kommt, dass bei dem Reichthum an Gefässen und bei deren allmähliger Degeneration und partieller Verlegung zeitweilige Schwankungen in der Compression und der Ernährung der vom Tumor bedrängten leitenden Elemente sehr wahrscheinlich sind, die dann zu einem Wechsel zwischen Besserung und Verschlimmerung der motorischen Innervation und der Reflexe geführt haben.

TAFELERKLÄRUNG.

Fig. 1. Rückenmark von vorn.
Fig. 2. Rückenmark von hinten.
Fig. 3–8. Rückenmarksquerschnitte. Weigertsche Markscheidenfärbung. S. Text.

FIG. 1 RÜCKENMARK VON VORN.
FIG. 2 RÜCKENMARK VON HINTEN.

Figs. 3–8 Rückenmarksquerschnitte.
Weigertsche Markscheidenfärbung.

A CASE OF MULTIPLE NEURO-FIBROMATA OF THE ULNAR NERVE.

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Surgical Report by Dr. Keen.

B. L., a laborer, from Northumberland, Pa., æt. forty-seven, first consulted me April 17th, 1899.

His father died of tuberculosis; mother, of unknown cause. Neither of them suffered from rheumatism. The patient has suffered for twenty years from rheumatism, especially in his knees. For many years past there have been some tender nodules in the palm of his left hand. He is doubtful if they are made worse by bad weather. At first they were painful only when compressed in handling a spade, axe, or any other such tool, but gradually the pain became continuous and kept him awake many nights. It was often so severe that he could scarcely refrain from outcries when it darted through his hand. As he expressed it, "The jumping toothache was n't in it compared to this pain."

In January, 1892, another surgeon removed two of these nodules. Stellate scars at each place mark the site of the wounds, which suppurated. No microscopical examination of the tumors seems to have been made. He has now nodules at the places marked *a* to *f*, Fig. 1. These existed at the time of the prior operation, but as they were not painful they were not removed. The one in the forearm marked *a* is painful only occasionally, when, by accident, pressure is applied to it. The others are all spontaneously painful.

For twenty years also he has had a fistula in ano, for which two

ineffectual operations have been done. Three openings about 3 cm. anterior to the anus, surrounded with a great deal of induration, were found.

Examination of the Sensation in the Left Hand of B. L., April 18, 1899, Just Before the Operation.

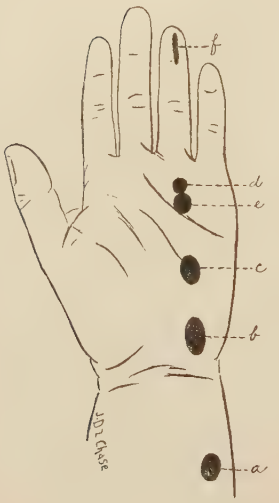

FIG. 1.

Diagram showing position of the tumors. Compare this with Fig. 4.

FIG. 2.

Diagrams showing the condition of sensation before the operation. The dark shading shows hyperæsthesia for all qualities, excepting cold, on the median side of ring finger. The lines represent the hypæsthetic area. On the back of the middle finger the hypæsthesia was uncertain.

Sensation was tested for heat, cold, pain, and touch.

The ulnar side of the left hand was *hyper*sensitive for all qualities except on the median side of the ring finger; here cold was not perceived quite so distinctly as in the corresponding portion of the right hand. The median side of the ring finger, palmar aspect, seemed to be somewhat *hyper*sensitive to all qualities except cold. The ulnar side of the middle finger, both palmar and dorsal aspects, showed diminished sensation. When the patient was touched over the scar on the palm of the left hand — the scar made by a previous operation on the ulnar nerve, — he perceived the sensation in another scar situated on the ring finger of the same hand. When he was touched over the scar in the ring finger, he located the sensation correctly. The hyperæsthesia was doubtless due to the irritation of the nerve fibres caused by the presence of the tumors. Pain prevented a firm grasp by the left hand.

OPERATION, April 18, 1899. — I first made an incision above the wrist and exposed the ulnar nerve (at *a*, Fig. 1), in which I found a fusiform expansion caused evidently by a mass in its interior. An incision made very carefully in the axis of the nerve enabled me to separate the fibres to each side, and with an Allis dissector to shell out from the interior of the nerve an oval tumor with a small filament 1 to 2 mm. in diameter at each pole of the tumor. It was quite a surprise

to me to find so large a tumor in view of its being so obscurely felt before operation. The same remark would hold true of all the others. A second incision was made at the eminence of the thumb (at *b*, Fig. 1), and a similar tumor again shelled out of the ulnar nerve. A third incision, just above the web between the ring and the little fingers (at *d*, *e*, Fig. 1), revealed two such tumors, and as I could now feel another tumor in the middle of the palm (*c*, Fig. 1), midway between the last two incisions, I made these incisions continuous, and shelled out all the tumors. On the pulp of the ring finger opposite the last interphalangeal joint (*f*, Fig. 1), two quite small tumors were found, and the entire nerve with these two tumors was removed (see *f*, Fig. 4). All the tumors were oval in shape, with a small filament at each pole (see Fig. 4).

The fistula was then operated upon.

He made an uninterrupted recovery without febrile reaction, and left the hospital on the twelfth day entirely well.

Examination of the Sensation in the Left Hand of B. L., April 27, 1899, Nine Days After the Operation.

FIG. 3.

Diagrams showing the condition of sensation after the operation. The dark shading represents the nearly anæsthetic area, the lines on the palmar surface represent the hypæsthetic area, and the dots the hyperæsthetic area. On the middle finger the hyperæsthesia was uncertain. On the back of the hand the lines represent the slightly hyperæsthetic area.

Sensation was tested for heat, cold, pain, and touch, and the limits of the areas corresponded for each form of sensation, *i. e.*, there was no dissociation of sensation. The palmar surface of the left ring finger was almost anæsthetic in all forms of sensation. The *ulnar* side of the middle finger was hypæsthetic, but the thumb side of the middle finger and the

whole of the little finger, and the ulnar side of the palm as high as the wrist, were slightly *hyper*æsthetic, except that the portion of the palm above the middle and ring fingers was *hyp*æsthetic; above the wrist disturbance of sensation was uncertain. The thumb and first finger exhibited normal sensation.

On the dorsal aspect the middle, ring, and little fingers were somewhat more sensitive than the corresponding fingers of the right hand. The thumb and first finger were normal. The back of the hand was also normal.

He writes me under date of November 14th that his present condition is as follows: He has no pain either in his hand or arm and has full use of the hand, but still suffers from rheumatism.

REMARKS. — In Dr. Spiller's report on such neuro-fibromata, most of the essential facts are stated, and many of the most important references are given and need not be recapitulated here. Operation is essential, for if left the tumor steadily increases in size. This results in constantly increasing pain, and by reason both of the pain and its consequent motor disturbances, the usefulness of the hand is impaired or even lost. Moreover, from the constant pain the patient becomes very irritable. Malignant degeneration in a nerve after neuro-fibromata have been removed, is by no means unknown, but the risk incurred by such an operation is justifiable in view of the symptoms produced by the tumors, and malignant degeneration may occur in these tumors even when no operation has been performed. In the present case the mode of operation was clearly indicated, and when the tumors are central in the nerve, as in this case, they should be shelled out. If they exist upon the side, they should be dissected carefully from the nerve, doing as little injury to its fibres as possible, yet removing as far as possible all the diseased tissue. Whether all diseased tissue has been removed is an extremely difficult thing to determine absolutely, for from each pole of the tumor the disease may pass a considerable distance up and down the nerve, or it may exist at some other part of the nerve without causing clinical manifestations. If the tumor is too large or too intimately connected with the nerve to allow of either of these methods of operation, two resources still remain; first, a resection of the entire nerve or amputation of the limb. The latter is not uncommonly necessary in the leg, because such tumors, in connection with the sciatic are apt to be very large and also to undergo malignant change. Resection of even so large a nerve as the sciatic is not always followed by total paralysis, and even should this follow, sensation and motion may occasionally be restored either through lateral paths or by a greater or less regeneration of the nerve. Such complete regeneration I have seen personally more than once in the inferior dental nerve.

One danger attends operation, especially if all the diseased tissue has not been removed, namely, a rapid and malignant recurrence.

FIG. 4.—Actual size of tumors removed. For position of the growths see Fig. 1.

Tumor *a*.—Almost symmetrically oval, measuring 10 x 9 mm. Tumor *b*.—Irregularly elongated, measuring 18 x 9 mm. Tumor *c*.—Measures 13 x 8 mm. Tumors *d* and *e*.—Two distinct growths, the longer one uppermost, measuring 6 x 4 mm; the smaller one measuring 4 x 3 mm. Tumor *f*.—Only a thickening of the nerve, presenting no definite tumor formation.

FIG. 5.—A section from the tumor *c*, Fig. 4, taken near the centre of the growth.

a.—Perineurium. *b*.—Loose connective tissue near the periphery of the growth. *c*.—Dense connective tissue, forming the centre of the tumor.

In Robert W. Smith's monograph, republished in 1898 by the New Sydenham Society, many admirable drawings of such tumors will be found.

Pathological Report by Dr. Spiller.

From the William Pepper Laboratory of Clinical Medicine, University of Pennsylvania. (Phœbe A. Hearst Foundation.)

The tumor designated in Fig. 4 as *c*, was studied microscopically. Sections made about half way between the two ends of the growth showed that the centre of the sections was formed by dense bands of connective tissue interwoven with one another and running in various directions. Most of these fibres had a transverse course, but some ran longitudinally, and were therefore cut transversely in transverse sections of the tumor. Numerous nuclei, deeply stained by Delafield's hematoxylin, were found mingled with the fibres, and most of these nuclei were elongated; some, however, were round. It is difficult to determine whether the latter were merely elongated nuclei, cut transversely, or were really round nuclei, but probably most of them were elongated nuclei. Towards the periphery of the growth the fibrous tissue was looser and contained many nuclei, and this looser fibrous tissue passed rather abruptly into the circular bands forming the perineurium (Fig. 5). Blood-vessels were rare in the centre of the tumor where the dense fibrous tissue was found, but were quite numerous in the looser tissue at the periphery. About some of the vessels at the periphery of the growth an area was found that showed a groundwork not staining well with ammonium carmine but containing numerous delicate wavy fibres arranged circularly about the vessel. By Weigert's hematoxylin method a few scattered nerve fibres were seen within the looser tissue at the periphery of the tumor, but none were found in the central dense fibrous formation. The perineurium was not much thickened. The proliferation of connective tissue evidently began in the endoneurium and in the centre of a nerve bundle. Nerve fibres were found in sections taken from each end of the tumor (Figs. 6 and 7), and it was evident from these and the sections from the middle of the tumor, that nerve fibres extended from one extremity of the tumor to the other, although they were not numerous.

The description of the tumor designated as *c* in Fig. 4 applies to the smaller growth designated by the letter *d* at the part marked by a horizontal line. In the tumor *d* the zone of looser fibrous tissue was between a dense central and a dense peripheral area. This small tumor also contained a few nerve fibres distinctly shown by Weigert's

hematoxylin method. In some of the nerve bundles removed—seen as a narrow band upon the tumor *d* in Fig. 4—and cut with the tumor *d*, the connective-tissue proliferation was seen in an early stage; the individual nerve fibres were separated a little more than normally by a slight excess of connective tissue, and in some places where the connective tissue was in still greater amount the nerve fibres appeared somewhat atrophied, as though they had suffered from the pressure of the proliferated tissue. In these nerve bundles the proliferation was distinctly seen to begin in the endoneurium. These nerve bundles were distinct from the tumor *d*, but formed an integral part of the tumor immediately adjoining, designated as *e* in Fig. 4. This was proof that the proliferation of the endoneurium was not confined to one or two nerve bundles within the ulnar nerve, and it would indicate that at one part of the nerve one bundle was affected, and at another part another bundle.

The piece of nerve, designated as *f* in Fig. 4, was found by microscopical sections to contain three nerve bundles presenting different degrees of thickening of the endoneurium, and the perineurium of these bundles was considerably thickened.

The connective tissue of the nerves in some persons exhibits a remarkable tendency to proliferation, and this proliferation may cause the condition known as generalized neuro-fibromatosis, in which multiple neuro-fibromata, multiple cutaneous fibromata, plexiform neuro-fibromata, elephantiasis of the skin, and pigment nævi occur.

To one unfamiliar with the literature, the classification of fibro-neuromata with some of the forms of generalized fibromatosis mentioned would seem unwarrantable, and a brief résumé of some of the more important papers is desirable for a proper understanding of this rather difficult subject, especially as most of the literature is in the German language.

The relation of these various pathological formations to one another has not been universally recognized. Virchow believed that a relation existed between the multiple cutaneous fibromata, the neuro-fibromata, and certain forms of congenital elephantiasis (Goldmann), and v. Recklinghausen [1] showed that the fibromata mollusca are fibromata developed on the cutaneous branches of nerves. Marie [2] states, in an excellent paper on this subject, that v. Recklinghausen's view is pretty generally accepted, but not by all. Marie does not include every fibroma molluscum under the generalized neuro-fibromatosis. In one of the two cases of generalized neuro-fibromatosis reported by him an

[1] v. Recklinghausen, *Ueber die multiplen Fibrome der Haut*, etc., 1882.

[2] Marie, *Leçons de clinique médicale*, Hotel Dieu, 1894–1895.

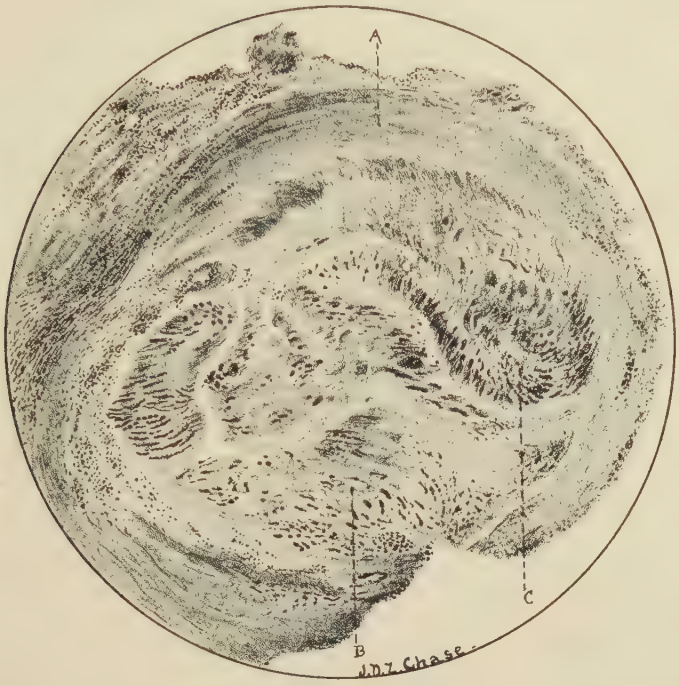

FIG. 6.—Transverse section from one extremity of tumor *c*, Fig. 4, showing the nerve fibres widely separated from one another by proliferated connective tissue.

A.—Perineurium. *B* and *C*.—Nerve fibres.

FIG. 7.—Longitudinal section of tumor *c*, Fig. 4, showing medullated nerve fibres (*A*) entering at one extremity of the growth and passing to the periphery of the tumor.

autopsy was obtained. He was unable to find any fibromata on the nerves, or to find nerve fibres in the cutaneous tumors removed from the cadaver. The case clinically was a typical one of generalized neuro-fibromatosis, and in two cutaneous tumors removed during the life of this patient a few nerve fibres were found. The absence of nerve fibres in the tumors removed from the cadaver of course does not prove that these tumors did not have their origin in the cutaneous nerves. The cutaneous nerves are small and may be destroyed in fibromatous thickening. The examination of the growths removed by Professor Keen has shown me that the nerve fibres within the tumor may be entirely destroyed in places by the proliferation of the connective tissue, and this is doubtless true of the nerve fibres in the fibromata of the skin.

While certain writers have contended that the multiple fibromata of the skin arise in the connective tissue of the cutaneous nerves, some make the connective tissue surrounding other structures—about the roots of the hair, glands and vessels—responsible for the proliferation. This question has not been positively decided. For example, Goldmann[1] reported a case of congenital plexiform neuroma (the first case in his paper) in which microscopically numerous fibromata were found in the skin, and all of these were in relation with cutaneous nerves. Apparent thickening of the adventitia of the vessels, of the hair sheaths, of the sweat and sebaceous glands, was in reality due to a fibromatosis of the nerves supplying and surrounding these structures. This case supports the view of the unity of the process in multiple cutaneous fibromata and multiple fibro-neuromata of the nerves. On the other hand, in a paper published by Finotti[2] three years later, different conclusions were reached. Finotti believed from his studies that the multiple fibromata did not originate exclusively in the nerves. The writers seem to agree that in structure the cutaneous fibromata and the fibro-neuromata of the nerves are very similar. The literature on this subject is given in a recent paper by Merken.[3]

The generalized neuro-fibromatosis is known also as v. Recklinghausen's disease and as elephantiasis nervorum. Under the latter term Hartmann[4] classes the neuro-fibromata of the nerves, the fibromata mollusca, cutaneous enlargements, and pigment spots,—all are manifestations of a congenital tendency of peripheral nerves to tumor-formation. The designation of elephantiasis neuromatodes is employed

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[1] Goldmann, *Beiträge zur klin. Chirurgie*, vol. x., 1893.

[2] Finotti, *Virchow's Archiv*, vol. cxliii., 1896, p. 133.

[3] P. Merken, *Wiener klin. Wochenschrift*, Nos. 32, 33, and 34, 1899.

[4] Hartmann, *Beiträge zur klin. Chirurgie*, vol xvii., p. 177.

by Scheven[1] to include plexiform neuromata, fibromata mollusca, and fibromatous thickening of nerve trunks.

The plexiform neuroma seems to be a part of generalized neurofibromatosis. P. Bruns[2] has had an unusual opportunity to study this form of growth,—as the study of eight cases of this rare tumor may well be considered unusual. He says that the Rankenneuroma (plexiform neuroma) is one of the forms of congenital elephantiasis—known also as the fibromatous diathesis—and is the result of fibromatous thickening of the nerves of a circumscribed territory. It differs only in form from the multiple fibromata of the skin and nerve trunks. This is shown by the congenital and occasionally hereditary tendency to the formation, the simultaneous appearance of the different forms in the same person, the same histological structure, etc. Bruns was able to collect in all, including his own, forty-two cases of plexiform neuroma from the literature (1891); in three of these heredity was observed through three generations; in the first two generations multiple cutaneous fibromata, multiple fibromata of the nerve trunks, and elephantiasis occurred, and in the third generation the plexiform neuroma appeared. Bruns, therefore, classes under elephantiasis the plexiform neuromata (Rankenneuroma), multiple cutaneous fibromata, multiple fibromata of the nerves, and colossal elephantiasis; and he shows that the disease may be hereditary, appearing in one form in one generation and in another in a following generation. The term elephantiasis neuromatodes seems to have originated with Bruns, but he distinguishes other forms: the elephantiasis teleangiectodes, and the elephantiasis lymphangiectodes, according as the blood- or lymph-vessels are involved.

The relation between multiple fibro-neuromata and elephantiasis was recognized in two cases by Jordan.[3] The vascular system was the source of a connective-tissue hyperplasia causing great thickening of the skin and subcutaneous tissue and the formation of tumors in the nerves and muscles. Fibromata mollusca were found in both cases. In one patient the right lower limb had a circumference of 75 cm. and the left 22 cm., and the right lower limb was greatly deformed. Jordan was unable to classify these cases under any of the three forms of elephantiasis (elephantiasisfibro matosa, teleangiectodes, neuromatodes) known to him. Macroscopically the condition was one of elephantiasis fibromatosa with multiple fibromata mollusca, multiple neuromata,

[1] Scheven, *Beiträge zur klin. Chirurgie*, vol. xvii., p. 157.

[2] P. Bruns, *Beiträge zur klin. Chirurgie*, vol. viii., p. 1.

[3] Jordan, *Beiträge zur pathologischen Anatomie und zur Allgemeinen Pathologie*, vol. viii.

and thickening of the large vessels; microscopically the vessels were found to be the structures in which the hyperplasia began, so that in both cases there was a combination of different forms of congenital elephantiasis. Jordan doubts whether cases of congenital elephantiasis in which only the skin and subcutaneous tissues are affected exist, *i. e.*, cases of solitary pachydermatocele.

Herczel[1] was able to observe that in a pachydermatocele the proliferation of the connective tissue originated in the thickened fibrous nerve bands; that it was a true elephantiasis neuromatodes.

The frequency of pigment nævi in generalized fibromatosis has been noted by many writers, and in a careful microscopical study Soldan[2] has recently shown that these pigment nævi (Pigmentmäler) are in the majority of cases the first recognizable signs of a fibromatous process of the connective tissue of the nerves. The different forms of neuro-fibromatosis are conditioned by the localization, anatomical relations, and energy of growth.

This brief summary of some of the most important writings on generalized neuro-fibromatosis is sufficient to show that we are justified in classing under one head the neuro-fibromata of the nerves, the cutaneous fibromata, the plexiform neuromata, certain forms of elephantiasis, and certain pigment nævi.

The causes of generalized neuro-fibromatosis are unknown. Reference has been made to the fact that heredity plays a rôle in some families, and in addition to the cases cited I may mention that Menke[3] observed neuromata in members of three generations, grandmother, mother, and son, and he says a heredity through three generations existed only in the cases cited by Herczel, Bruns, and Czerny. Most writers agree that the condition is not usually an acquired one. The tendency exists from the birth of the person, although the proliferation of connective tissue may not be observed until comparatively late in life. Trauma may in some cases cause the manifestation of a latent tendency in nerves.

Another important question that demands attention is in relation to the tendency of these fibromata to malignant degeneration. Several investigators have shown that this danger is not an imaginary one. Goldmann (*l. c.*) demonstrated by one of his cases that an apparently benign neuro-fibroma may undergo malignant change, or, better stated, present a malignant course, and he quotes a number of similar cases. According to him, this malignancy is not a change in the character of

[1] Herczel, *Ziegler's Beiträge*, vol. viii.

[2] Soldan, *Archiv für klin. Chirurgie*, vol. lix., No. 2, p. 261.

[3] Menke, *Berliner klin. Wochenschrift*, No. 44, Oct. 31, 1898, p. 974.

the tumor, but is due to the fact that a sarcoma of the nervous system may occasionally show a slow growth for a long time.

According to Finotti (*l. c.*), numerous observations have demonstrated that solitary plexiform (Herczel) and multiple neuromata (Genersick, Czerny, Winiwarter, Westphalen) have a great tendency to change into sarcomatous tissue. His words are "Umwandlung in Sarcomgewebe." Distinct clinical differences between primary and secondary sarcoma—that is, neuro-fibromata that have undergone sarcomatous change and those that are sarcomatous from the beginning—do not exist, according to Finotti, at least not in the majority of the cases.

Trauma may be the cause of this malignant degeneration, but in some cases no cause can be demonstrated (Garrè, Hartmann, and others). Hartmann states that one of a number of neuromata may increase rapidly in size, and when it is removed by operation another tumor may develop rapidly in the same nerve trunk at the site of the former growth,—more frequently, however, in another nerve territory. The second tumor is usually more malignant than the first; it involves adjoining tissue, and comparatively late metastasis occurs, causing the death of the patient. Garrè[1] also noted the increased malignancy of the process after operation. Hartmann[2] reports a case which he says showed well the transformation of a fibroma of the nerve into a sarcoma, and he confirms the experience of others that the rapid increase in a neuroma, the occurrence of neuralgic pain, and sarcomatous degeneration occur at the same time. He refers also to the fact that in ten of the seventeen cases of malignant degeneration in fibro-neuromata mentioned by Garrè, death was due to a return of the tumor.

Four cases of general neuro-fibromatosis, with multiple neuro-fibromata, were observed by Thomson.[3] In the first of these, one of the tumors underwent sarcomatous change, with general dissemination of sarcoma, and death after attempted removal; in the second also, after operative intervention, malignant change occurred. Scheven (*l. c.*) also refers to the pronounced tendency of the elephantiasis neuromatodes congenita to sarcomatous change, and to the fact that this tendency remains in the fibromatous nerves after a tumor that has undergone a malignant change has been removed. The surgeon can do nothing more than remove the malignant tumor; the tendency to degeneration remains in the widely-developed pathological tissue. Scheven reports also a case of malignant degeneration in elephantiasis neuromatodes, and refers to the fact that in Finotti's case the

[1] Garrè, *Beiträge zur klin. Chirurgie*, vol. ix., p. 465.

[2] Hartmann, *Beiträge zur klin. Chirurgie*, vol. xvii., p. 177.

[3] Thomson, *British Med. Journal*, Oct. 10, 1896, p. 1024.

sarcomatous transformation in the neuroma could be demonstrated microscopically.

We owe to Garrè's (*l. c.*) investigations the knowledge of the frequency of the degenerative change in neuro-fibromata. Garrè was able to collect sixteen cases from the literature—seventeen with one of his own—in which sarcomatous degeneration in congenital neuromatosis had occurred. There are, of course, many more cases of malignant tumors of nerves not the result of degeneration of a fibroneuroma. In these seventeen cases, those of sarcoma arising in cutaneous fibromata are not included. Garrè showed that in an eighth of all cases of supposed benign neuro-fibromata this sarcomatous change occurs.

It is important to know what constitutes a sarcomatous degeneration in a neuro-fibroma. Rapidity of growth is suspicious, according to Garrè, but is not always reliable; large size of the growth is not a positive sign, and even histologically the transformation of a benign fibroma into a sarcoma may be difficult to determine. The greater or smaller number of tumor cells is the determinative factor, but there are cases in which the diagnosis between fibroma and sarcoma cannot be made with certainty, and Garrè says that there are transitional forms between the neuro-fibroma and the sarcoma, and that these cannot be properly classed clinically or histologically. Paræsthesia, paresis, neuralgic pain, etc., are important in diagnosticating early and clinically the sarcomatous degeneration of a neuro-fibroma. The malignant change causes rapid destruction of the nerve fibres within the tumor, with the production of disturbances in motility and sensation.

It seems to me a broad and proper view to regard such neuro-fibromata as were removed by Professor Keen as an incomplete manifestation of generalized neuro-fibromatosis, although the process was confined to one nerve, the ulnar. The fibromatosis does not differ from that occurring in cases with more extensive clinical manifestations, and the limitation of the process so far is no proof that later we shall be unable to trace evidences of a more general fibromatous change. A number of cases have shown that the fibromatosis of nerves may remain undetected until the patient is well advanced in years. We understand likewise that Professor Keen's patient is exposed to the danger of malignant growth at any time.

The location of the tumors in Professor Keen's patient is especially interesting. Garrè, in speaking of his case, says that the presence of multiple small fibromata in the skin of the sole of the foot was very remarkable, and refers to the fact that v. Recklinghausen emphasized the immunity of this part and of the palm of the hand. Marie (*l. c.*)

likewise says that in generalized neuro-fibromatosis the tumors are not usually found in the hands and feet.

It is curious that only certain nerve bundles of the ulnar nerve were affected in Professor Keen's patient, but such a condition is well known. Goldmann (*l. c.*) says it is difficult to understand why in the same nerve trunk, even in the same bundle, the proliferation may involve only certain groups of fibres.

Bowlby[1] describes a specimen of multiple fibromata on a single nerve (posterior tibial) seen in the museum of the Middlesex Hospital. No tumors were found on any other nerve. This seems to have been the only case of the kind which had come under his observation when he wrote his book. A similar case was published by J. K. Mitchell,[2] and W. J. Taylor operated on a patient with neuro-fibromata of the foot, probably confined to a single nerve. As a contrast to Professor Keen's case and to Bowlby's, I may mention that Smith reported one case in which the total number of neuro-fibromata existing upon the nerves removed from the body exceeded 800, and another in which upwards of 1400 neuromatous tumors were removed with the nerves, and he felt that he was not exaggerating in stating that this patient must have had at least 2000 tumors. This work of Smith was originally published in the form of a very limited edition fifty years ago, but it was deemed of sufficient importance to be reprinted in 1898 by the New Sydenham Society. Some of the plates present nerves covered thickly with fibro-neuromata, but no neuromata shelled out of a nerve, as in Professor Keen's case, are pictured in this atlas. Indeed, Smith[3] stated that the results had not afforded much encouragement to the practice of dissecting out the tumor from the branches of the nerve among which it was entangled. According to Smith, there are few affections more rare than neuro-fibroma.

Smith was not able to trace nerve fibres through a neuro-fibroma, although in a few instances he observed some nervous filaments entering the superior extremity of the tumor. I have been able to detect the presence of nerve fibres in both extremities and also in the centre of one of the tumors (*c*, Fig. 4) removed by Professor Keen, and my success was probably due to the fact that I had Weigert's hematoxylin stain at my command. In the smaller tumor (*d*), nerve fibres were also seen within the tumor. This method was unknown when Smith wrote his treatise.

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[1] Bowlby, *Injuries and Diseases of Nerves and their Surgical Treatment*, p. 493.

[2] J. K. Mitchell, *The University Med. Magazine*, 1897, Nov.

[3] Smith, *A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma.* The New Sydenham Society, 1898.

It is a merciful provision that the multiple neuro-fibromata are usually painless, and it seems extraordinary in contrast that the solitary tumor is often painful.

Many investigators have observed that the proliferation begins in the endoneurium, as it did in Professor Keen's case. It is due to this mode of origin that these neuro-fibromata are elongated with their long axes parallel to the nerve. The perineurium offers a certain amount of resistance, especially as it often becomes thickened simultaneously with the growth of the tumor, and the fibroma grows especially in the direction of least resistance. In the tumor designated as *c* in the drawing (Fig. 4), the perineurium formed a sheath, surrounding on all sides the proliferated endoneurium, and in the centre of the tumor no nerve fibres could be detected, except at the periphery. This proliferation of the endoneurium was evident also in the tumor *d* (Fig. 4), and in small nerve bundles, adjoining the tumor *d* and removed with it, the proliferation of the endoneurium could be detected in its early stages. Neuro-fibromata do not always originate in the endoneurium. In a case reported by Finotti, for example, the fibroma began in the epineurium and was adherent to the nerve.

The entire nerve on which the neuroma is formed may lie in the centre of the tumor or surround the tumor as a sheath. According to L. Bruns,[1] the nerve fibres caught within the neuro-fibroma show a remarkable resistance to degenerative processes, which explains the absence or mildness of the clinical symptoms in many cases. I am unable to fully confirm this statement from the examination of the tumors removed by Professor Keen. In two tumors, *c* and *d*, I found the nerve fibres within the tumor entirely destroyed, except at the periphery of the tumors, while in the piece of nerve marked *f*, in which the proliferation was not excessive, the nerve fibres were quite well preserved. I would prefer to explain the absence or mildness of the clinical symptoms by the slowness of the process, by the fact that only here and there a nerve bundle is attacked, and that many fibres — the majority in fact — remain intact and do not lose their function although considerably compressed, as the process is slow enough to allow the nerve fibres to become accustomed to the pressure.

[1] L. Bruns, *Die Geschwülste des Nervensystems.*

SOME BACTERIOLOGICAL POISONS IN MILK AND MILK PRODUCTS.

By VICTOR C. VAUGHAN and JULIAN T. McCLYMONDS.

IN 1884, one of us obtained from poisonous cheese a basic, crystalline substance which caused in man and the lower animals nausea, vomiting, and purging. The amount of this substance obtained was very small and not sufficient to permit a thorough study of its properties or a determination of its ultimate composition. From 16 kilos. of one cheese about 0.5 gram of the crystals was separated, while the same quantity of another poisonous cheese furnished less than 0.1 gram. It was not supposed, however, that all the poison in the cheese was obtained. To this substance the name tyrotoxicon (cheese poison), which had already been used to designate the undiscovered active agent in poisonous cheese, was given. The only definite statements that could be made about this substance at that time were:

(1) It was obtained from certain samples of cheese, the eating of which had caused nausea, vomiting, and purging in about three hundred persons, and the extracted crystals induced the same symptoms. (2) It is a crystalline substance. The crystals obtained from some of the samples of cheese were plainly visible to the unaided eye. (3) It is freely soluble in both water and absolute alcohol. (4) In aqueous solution it is decomposed at 100° C.[1]

The study of the above-mentioned poison was continued, and several investigators succeeded in isolating it and contributed valuable facts concerning its development in milk and milk products.[2]

It was at first supposed that this basic poison is the active agent in most samples of poisonous milk and milk products, but larger experience has shown that this is not true. Many samples of supposedly poisonous cheese were sent to the Hygienic Laboratory of the University of Michigan, in which tyrotoxicon could not be found, and

[1] "Ein Ptomaïn aus giftigen Käse." *Zeitschrift f. physiologische Chemie*, Bd. x., S. 146.

[2] "Ueber die Anwesenheit von Tyrotoxicon in giftigen Eis und giftiger Milch und seine wahrscheinliche Beziehung zur Cholera Infantum." *Archiv f. Hygiene*, Bd. vii., S. 420.

which were consequently pronounced not to be responsible for the ill effects attributed to them. After some time, samples of this kind became so numerous and, in some instances at least, the evidence of their poisonous properties was so convincing that, when tyrotoxicon could not be found, other poisons were sought. Some of these caused vomiting and purging in cats and dogs to which the cheese was fed. In this way the evidence that these samples had been the actual cause of the sickness among the people who had eaten of them was confirmed by the experiments upon the animals, but inasmuch as the poison could not be isolated, the following report was made: "The poisonous character of the cheese has been proven by experiments upon animals, but the nature of the poison has not been discovered. Tyrotoxicon is not present." One sample of this cheese was found by Dr. Novy to be quite poisonous. A piece was covered with absolute alcohol for some weeks, after which the alcohol was removed and 100 grams fed to a young dog, causing its death within a few hours. Sterilized milk to which a small bit of the cheese had been added, after standing in the incubator at 35° for twenty-four hours, became so poisonous that 100 c. c. of it introduced into the stomach of a full-grown cat caused death.

In a second class of samples of cheese and milk the poisonous character of the food was not confirmed by direct feeding. Cats, rats, and dogs were fed exclusively and for long periods of time on these articles without any appreciable effect. After examining samples of this class, the report made usually read something like the following: "Animals fed upon the suspected articles were not affected. Tyrotoxicon could not be found. The sickness observed in your patients was probably due to some other cause."

A closer study of samples of poisonous milk and milk products demonstrated that these articles of food sometimes contain soluble poisonous proteids. At least one of these belongs to the so-called poisonous albumins. It is freely soluble in water, from which it is not precipitated by heat or nitric acid, singly or combined. Its aqueous solutions respond to the biuret test. It is not precipitated by saturations with sodium sulphate, nor by a current of carbonic acid gas; therefore it is not a globulin. It is precipitated by saturation with ammonium sulphate; and this fact distinguishes it from the peptones.[1]

In 1895, one of us, assisted by Perkins, undertook a systematic study of samples of cheese that had seriously poisoned many people. The toxicogenic germ in these articles of food was isolated and studied. This bacillus resembles somewhat the bacterium coli commune, but differs from the same in the following important particulars:

[1] *Medical and Surgical Reporter*, vol. lxiii., 1890.

(1) The cheese bacillus does not furnish cultures that give the indol reaction. (2) Both coagulate milk, but the cheese germ acts in this particular much more rapidly than the colon bacillus. It may be stated that in these experiments colon bacilli from two sources were employed: one culture had been in the laboratory for some years and had been brought by one of us from the Hygienic Institute in Berlin, while the other was freshly obtained from normal fæces for purposes of this experiment. (3) Milk cultures of the new bacillus have a pleasant odor of butyric ether, not possessed by similar cultures of the colon germ. (4) The new germ grows abundantly on carrots, forming a raised, creamy layer, and giving off a sour odor; while the colon bacillus grows much less vigorously on this medium and gives off no such odor. On turnips, the new germ grows vigorously, forming a thick grayish layer, and this also develops a sour odor; while on the same medium, the colon bacillus develops relatively feebly. On bananas, onions, parsnips, and apples, the new bacillus grows much more abundantly than does the colon bacillus. (5) Milk colored with rosolic acid is decolorized much more quickly by this bacillus than by the colon germ.

Milk cultures of this germ elaborate a chemical poison which is freely soluble in absolute alcohol, and in this respect resembles tyrotoxicon, but that it is not tyrotoxicon, we were able to demonstrate both chemically and physiologically. From a filtered culture, after concentration in vacuo under 40° and after being rendered alkaline with either a fixed or a volatile alkali, the poison is not removed by ether. This distinguishes the new poison chemically from tyrotoxicon. Physiologically this substance is distinguished from tyrotoxicon by the more pronounced effect of the former on the heart, in which it resembles muscarin or neurin more closely than it does tyrotoxicon. Pathologically the two are unlike, inasmuch as the product of the new bacillus produces marked congestion about the point of injection, or in the peritoneum when thrown into the abdominal cavity. Moreover, the intestinal constriction, which was so constantly observed in animals poisoned with tyrotoxicon, was not once seen in experiments with this germ, although it was carefully looked for in each of the more than two hundred animals used in the study.[1]

The above résumé briefly outlines the work done in investigating the bacterial poisons found in milk and milk products in the Hygienic Laboratory of the University of Michigan up to the beginning of our joint researches. We began our experiments in 1897, and continued

[1] "Ein in Eiscreme und Käse gefundener giftproducirender Bacillus." *Archiv f. Hygiene*, Bd. xxvii., S. 308.

them without prolonged interruption until June, 1898. Other duties have up to the present prevented us from preparing our results for publication, and, in doing so now, we will summarize the voluminous records of our protocol book.

The special object in view was to ascertain to what extent ordinary cheese is infected with toxicogenic bacteria, what the source of these harmful organisms is, and what are the chemical poisons elaborated by them. We obtained sixty-five samples of cheese from as many manufactures and submitted these to an examination, the details of which may be stated as follows: On the receipt of each sample a record was made of the place and date of manufacture (when these could be ascertained), the kind of cheese, odor, taste, reaction of the interior to litmus, and appearance and number of cavities, with any other facts of interest known. Beef-tea tubes were inoculated with small bits taken from the interior of the samples with a sterilized platinum loop. These tubes were kept for twenty-four hours in the incubator at 38°. At the expiration of this time gelatine and agar plates were made, and a rabbit, a white rat, and a guinea-pig each received in the abdominal cavity 2 c. c. of the beef-tea culture. From the ordinary plates each germ was obtained and grown in pure culture. From each animal that died from the effects of the inoculation, plates were made and from these pure cultures were obtained. However, we never found more than one germ in plates made from blood taken from the heart of the dead animal. The micro-organisms found on the plates developed from the blood of the heart of the animals was compared with those developed on the plates made directly from the cheese. It will be seen that in these experiments the animals served as living plates and enabled us to separate the toxicogenic from the non-toxicogenic bacteria. After this had been done, the toxicogenic organism was studied in pure culture. Each germ obtained from the animal plates was thoroughly studied in the following particulars: (1) The growth and appearance of colonies on gelatine plates. (2) The growth on both gelatine and agar, both in stab and smear cultures. (3) Growth on potato. (4) Growth in beef-tea, both aërobically and anaërobically. (5) Growth on glucose agar. (6) Growth in milk, with especial attention to time necessary to cause thorough coagulation. (7) The presence or absence of the indol reaction in beef peptone cultures and the time necessary to develop the test. (8) Behavior with ordinary stains. (9) Appearance in hanging drop, with special reference to motility. (10) The effect upon animals of different amounts of pure cultures. In this study special attention was given to the macroscopic appearance observed after death. (11) The effect upon animals of sterilized cultures.

The space allotted us for this communication will not permit anything more than the following brief statement of the results obtained:

Of the 65 samples examined, 49 were the ordinary American green cheese. These were made in Michigan, Wisconsin, Illinois, New York, and Canada. Eight of the 49 samples were sent us because persons eating them had suffered from nausea, vomiting, and purging. Every one of the 49 samples of American green cheese furnished cultures which killed white rats, guinea-pigs, and rabbits. The toxicogenic germ in all of these samples belongs to the colon group. While the organisms obtained from different samples show minor variations in cultural properties, all agree in the following:

(1) Beef peptone cultures four days old give the indol test. The rapidity with which indol is developed varies considerably in cultures made from different samples of cheese. In some the reaction is well marked at the expiration of 24 hours, while in others, only a slight coloration results after 72 hours.

(2) All the cultures coagulate milk within 72 hours; some, completely within 24 hours.

(3) All ferment glucose cultures. In this particular, also, bacteria from different samples show wide variations in the energy with which they act.

(4) None of the toxicogenic bacteria found in the samples of American green cheese liquefy gelatine.

Most of the germs as seen in hanging-drop show only a vibratory motion; a few are slowly motile, and still fewer are actively motile. The colonies on gelatine plates show considerable uniformity, being for the most part brownish disks; some of the colonies form rosettes. The surface growths show frequent irregularities. In form, these toxicogenic bacteria consist of short rods with rounded ends. In length they average from two to three μ, but in many instances the length is so slightly in excess of the breadth that the appearance is very similar to that of micrococci. The pathogenic properties of the germs obtained from American green cheese are shown by the following quotations taken from the records of hundreds of experiments in our protocol book:

"November 11, 1897: Rabbit No. 2 received intra-abdominally at 5 P.M. 2 c. c. of a beef-tea culture from cheese No. 2. November 12th: found dead at 7:30 A.M. The subcutaneous tissue over the abdomen is markedly congested. The abdominal cavity contains a bloody fluid in which short bacilli with rounded ends are found in pure culture. The peritoneum is inflamed. The capsules of the kidneys show hemorrhagic spots. The spleen is normal; the liver pale; the heart is in diastole and filled with clotted blood.

"November 12, 1897: Rabbit No. 7 received intra-abdominally at 11:15 A.M. 2½ c. c.

of the fluid taken from the abdominal cavity of rabbit No. 2; died at 5:15 P.M. of the same day. There is slight subcutaneous congestion over the abdomen. The mesentery shows numerous hemorrhagic extravasations and the serous coat of the stomach and small intestines is dotted with hemorrhagic spots. The peritoneal cavity contains a small amount of fluid containing the cheese bacillus in pure cultures.

"November 12, 1897: Rabbit No. 14 received at 5:45 P.M. intra-abdominally 1 c. c. of fluid from rabbit No. 7. This animal was found dead at 9:00 A.M., November 13th. The subcutaneous tissue over the abdomen is greatly congested. The peritoneum is highly inflamed and the serous coat of the large intestines shows numerous hemorrhagic spots. The omentum also is hemorrhagic. There are small amounts of bloody fluid in both the abdominal and thoracic cavities. The heart is in diastole.

"November 13, 1897: Rabbit No. 17 received intra-abdominally at 11:00 A.M. ½ c. c. of fluid from the abdominal cavity of No. 14; found dead at 7:00 A.M., November 14th. When the abdomen is opened the small intestines look like a gelatinous mass. The peritoneal coat is congested and shows numerous pin-point hemorrhagic spots. Large extravasations are found in the intestinal loops along the attachment of the mesentery. The omentum shows numerous hemorrhagic spots which are largest and most numerous along the free-border. The pericardium also shows hemorrhagic spots. There is a small amount of fluid in the abdominal cavity.

"November 14, 1897: Rabbit No. 18 received at 4:00 P.M. intra-abdominally ¼ c. c. of fluid from the abdominal cavity of No. 17; found dead at 7:30 A.M. November 19th. Blood had flowed from the nose and mouth before death. The subcutaneous tissue over the abdomen is normal. The serous coat of the large intestine is highly congested and shows numerous hemorrhagic spots. The abdominal cavity contains 9 c. c. of bloody fluid."

From our studies, as already outlined, we conclude that the colon bacillus is present in practically all American green cheese. At times, other more active germs find their way into this article of food, but the colon bacillus is constantly present.

Having observed in several outbreaks of food-poisoning that the administration of opiates seemed to lessen the chance of recovery, we concluded that it might be of interest to try some experiments upon animals. The following illustrations from the protocol book show the method and the results:

"Guinea-pig No. 77 had at 5 P.M., February 20, 1898, ¼ grain of morphia sulphate hypodermatically. This animal continued well.

"Guinea-pig No. 78 had intra-abdominally at 5 P.M., February 28, 1898, ½ c. c. of a beef-tea culture of the cheese germ twenty-four hours old. This animal continued well.

"Guinea-pig No. 79 had at 5 P.M., February 28, 1898, intra-abdominally ½ c. c. of the same culture as No. 78 received, and immediately afterwards ¼ grain of morphia sulphate was injected hypodermatically. This animal was found dead at 7:30 A.M., March 1, 1898. The subcutaneous tissue was congested. The omentum was matted and hemorrhagic. The abdominal cavity contained 8 c. c. of fluid.

"Guinea-pig No. 80 had intra-abdominally at 5 P.M., February 28, 1898, ¼ c. c. of the culture given to Nos. 78 and 79. This animal continued well.

"Guinea-pig No. 81 had intra-abdominally at 5 P.M., February 28, 1898, ¼ c. c. of the cheese-germ culture, and immediately afterwards ¼ grain of morphia sulphate was injected hypodermatically. This animal was found dead at 7:00 A.M., March —, 1898. Subcutaneous congestion was marked. The omentum was matted and hemorrhagic, and the abdominal cavity contained 4 c. c. of fluid.

The above experiments were repeated several times without any variation in result. It is possible that the administration of opiates in appendicitis and other conditions in which there may be colon infection may not be altogether harmless.

The following extract from our protocol book illustrates our experiments made for the purpose of studying the chemical poison of the colon germ found in American green cheese :

" Ten Roux flasks properly filled with agar were inoculated with the cheese bacillus and allowed to stand one month at room temperature. At the expiration of this time the germ layers were detached from the agar by careful agitation with sterilized water. The total amount of water used in removing the germ from the ten flasks was 170 c. c. The moist germ mass was placed in a centrifuge which was kept going at about seventeen hundred revolutions per minute for twenty-four hours. At the end of this time the supernatant fluid was removed and filtered through porcelain in order to remove a few germs that remained in suspension. After filtration it was found to be a clear, yellowish fluid, slightly alkaline in reaction and inert in quantities of 5 c. c. given subcutaneously to rabbits and guinea-pigs. Even when dropped into absolute alcohol no precipitate occurred. This fluid apparently contains nothing but traces of coloring matter and alkali extracted from the agar. We conclude that our cheese germ forms, at least on agar, no soluble poison.

" The bacterial cells were thoroughly dried in vacuo over sulphuric acid, then rubbed up into a fine powder in a mortar. From the ten Roux flasks, we obtained 1.34 grams of powdered germ, which contained only traces of agar. One tenth of a gram of this powder was suspended in 10 c. c. of water and steamed for twenty minutes. This formed a milky-white substance. One c. c. of this, containing 0.01 of the powdered germ, was thrown into the abdominal cavity of a guinea-pig (No. 106) weighing 450 grams. The animal died in about four hours. The omentum and mesentery were congested and the abdominal cavity contained 5 c. c. of a straw-colored, sterile fluid.

" Prolonged digestion of the steamed germ with pepsin and hydrochloric acid does not lessen its poisonous properties. Indeed, prolonged steaming followed by several days' exposure to artificial gastric juice does not break up all the bacterial cells, as can be seen after staining and mounting."

It follows from these experiments that the chemical poison of this germ is contained within the bacterial cell. The fact that the colon bacillus does not form soluble poisons seems to explain how we are able to carry this germ in our intestine ordinarily without harm ; while,

on the other hand, the highly irritant action of this poison throws light upon the pathology of peritoneal inflammations. It of course remains to be demonstrated that all varieties of the colon bacillus produce the same poison or closely allied bodies. However, that this is the case is more than probable.

The space allotted us in this publication will not permit further discussion of this subject or a statement of the results obtained in the bacteriological examination of other kinds of cheese.

A CASE OF OTITIC BRAIN ABSCESS IN A BOY OF FIVE YEARS; OPERATION; RECOVERY.

By E. GRUENING, M.D.

THE number of recorded cases of otitic brain abscess terminating in recovery is small. There is a want of more literature, and the publication of a new and sufficiently well observed case may prove useful. The brain abscess here described developed in the course of an acute inflammation of the middle ear without perforation of the drumhead. At an early stage of the disease the mastoid became involved, as was shown by the formation of a retroauricular abscess. The boy, the son of a physician, was taken to the Mt. Sinai Hospital to be operated upon for the mastoid disease. Symptoms pointing to the brain were absent, and the existence of disease of that organ was not suspected. In the course of the operation, the dura overlying the antral roof was found to be discolored, softened, and fistulous, and the implication of the brain seemed evident. At that time the father did not permit me to follow up the fistulous tract into the brain, but later, when the symptoms of increased intracranial pressure became manifest, he consented to have the brain explored. By means of a bone forceps I removed the lower part of the squama, together with the roofs of the osseous meatus, antrum, and tympanum, and exposed a large area of the under surface and a narrow strip of the outer surface of the temporal lobe. The use of the trephine on the outer surface of the squama at so great a distance from the seat of disease was not indicated in this case. My observations agree with those of Körner and Jansen, who hold that cerebral abscess of otitic origin is generally found in the immediate vicinity of the primary bone trouble. This case is an additional proof of the correctness of this view inasmuch as the brain abscess lay so near the diseased spot in the bone that it could be reached by the shortest route. The opening of the abscess on the under surface of the brain facilitated the evacuation of pus and secured the best conditions for drainage. At no time was the abscess cavity irrigated; it was loosely packed with moist iodoform

gauze, and the packing was changed every day. The favorable termination of the case is probably due to the following factors:

1. The timely recognition of the abscess.
2. The absence of complications.
3. The situation of the abscess opening on the under surface of the brain.
4. The avoidance of irrigating fluids in the abscess cavity.

The chart registering pulse, temperature, and respiration is appended.

Patient W. A., five years old.

Previous History. — Three years ago had diphtheria, measles, and scarlatina. No discharge from ears.

Present History. — Fourteen days ago began to complain of slight pain in right ear, which disappeared after a few days. About ten days ago commenced to have fever — 100°–102.8° — (at same time two other members of family had malaria, and it was first thought that boy suffered from it also). Fever very irregular, on some days temperature being normal. Appetite always good. No chill, as far as known. About five or six days ago ear began to stand out from head, and soon after mastoid area was œdematous. No great tenderness. Few days ago fluctuation determined.

Status Præsens. — Right Ear: Drumhead bulging and red, especially above — *i. e.*, in Schrapnell's membrane; posterior and upper wall of canal swollen. Auricle stands off. Behind it, the skin over mastoid is partly covered with ichthyol and collodion. This being removed, distinct fluctuation. Left Ear: Schrapnell's membrane also reddened; vibrating membrane normal.

Pulse 120, temperature 101.5°, respirations 26.

May 3d. — Operation by Dr. Gruening. Ether anæsthesia.

Paracentesis of membrana tympani performed. No pus, but considerable bleeding. Culture taken of blood showed streptococcus longus. External auditory canal then packed, and incision made behind ear from linea temporalis to apex mastoid. As soon as skin and soft parts were cut through, considerable pus, evidently under tension, escaped; culture taken of this pus showed streptococcus longus. Periosteum reflected. Bone externally showed no evidence of perforation or disease. Bone over antrum attacked with gouge and mallet; softened carious bone soon encountered. Entire mastoid process was full of granulation tissue and carious bone. Antrum was cleaned out, and incus, which was loose in cavity and carious, was removed. The roof of the antrum was absent and the dura overlying it was soft and discolored, and at one point showed a fistulous opening. A packing was then placed against the dura, and also into the antrum, which had been thoroughly cleaned out. Sinus was then exposed and found normal, though bone all around it was carious. Diseased bone removed, also entire apex; wound packed without any sutures being introduced. At end of operation patient's general condition was good.

Temperature in P.M 103°, pulse 130, respirations 29; vomited several times.

May 4th. — In A.M., pulse 120–130, temperature 102°–104.6°, lowest temperature being at noon. About this time slight twitching of left arm noticed, sensorium otherwise good. Pulse and temperature fell gradually; at 10 P.M., pulse 114, respirations 24, temperature 101.2°. Patient takes moderate nourishment and no longer vomits. Wound dressed under chloroform. No change. Clean iodoform packings introduced, and wet dressing applied. General condition of patient quite good.

May 5th. — Dressed. Wound clean. Some softened tissue removed, and culture made of it. Wound sprayed with 1% carbolic and dressed; iodoform packings washed out in boracic acid, and wet dressing applied. General condition of patient good. Piece of

supposed brain tissue put into alcohol, and sent to pathologist, who reported it to be granulation tissue. Pulse, respiration, and temperature, normal.

May 6th. — Wound treated as above, and then dressed dry. Pulsation can be detected at bottom of wound.

May 7th. — Gradual rise of temperature to 101°. Wound therefore dressed wet. Dura beginning to be covered with granulation tissue.

May 8th. — Highest temperature 100°. Wound sprayed with carbolic ; dressed wet.

May 9th. — Temperature practically normal ; dressed dry ; on fluid diet. Fundus of eyes normal.

May 10th. — Pulse, respiration, and temperature, normal at 7 A.M.; rose gradually, and at 4 P.M., temperature 103.8°. At 9 P.M., pulse 134, respirations 34, temperature 105°. Patient vomited at 5 and 11 P.M. Patient put back on fluid diet. Free discharge of pus seen for the first time from external auditory canal. Also moderate discharge from wound, which looks very healthy. Canal dried and swabbed with 1 % carbolic ; wound sprayed with 1 % carbolic, packed with gauze, and a liberal wet 1 % carbolic dressing applied. This P.M., ¼ grain doses of calomel every fifteen minutes for four doses.

May 11th. — Several large movements to-day. This A.M., pulse 120, respirations 26, temperature 102.2°, fell gradually till at 9 P.M. pulse 98, respirations 26, temperature 100°. Dressed to-day in same manner as yesterday.

May 12th. — Temperature has continued to remain down. Dressed. Free discharge from wound ; none from ear. Wound filled with healthy granulations. Drain put into ear. Wound packed. Dressed with 1 % carbolic.

May 13th. — Free discharge from ear. Otherwise condition and treatment same.

May 14th. — Highest temperature 100°. Drain in ear changed night and morning. Mastoid wound not dressed.

May 16th. — Until to-day temperature had been normal, pulse 102–96, respirations 24. To-day it rose to 101.6°, pulse 114, respirations 26. Child more restless and appetite not so good. Wound covered with granulation tissue. Free discharge from ear. No sign o retention.

May 17th. — Temperature rose steadily until at 3 P.M. it was 104°, pulse 122, respirations 26. Child has refused nourishment and become drowsy. Wound exposed, and probe directed into region of tegmen tympani. Liberated about one drachm of thick pus. Child was at once taken to operating room, 5 P.M. Ether administered, and operation performed by Dr. Gruening.

Operation.—Granulation tissue was first removed, then wound was enlarged anteriorly above ear, so that squamous portion of temporal bone was well exposed. The part of the dura, which had been found softened at the previous operation was laid bare, and the bone around it removed to a moderate extent. A knife was then passed through the dura at this site, and two drachms of thick pus and necrotic brain tissue escaped. The opening was enlarged by dressing forceps, and the finger introduced into a cavity about three-fourths of an inch in diameter. This was then packed with iodoform gauze, as was the rest of the wound, and a wet dressing applied. The anterior part of the scalp wound was sutured. The brain abscess cavity was not irrigated. The malleus removed early in the operation. Previous to operation, ophthalmoscopic examination made with negative result. Spread and cultures from abscess pus showed streptococci.

May 18th. — Slept some during the night. At 2 A.M., temperature 101.9°, pulse 122, rose to 104.2° at 4 P.M., when wound was dressed. There was very little discharge from the wound. Packing in abscess cavity was replaced by Robert Morris protective drain. Wet dressing of 1 % carbolic. Despite the temperature, child was brighter, and moved his head about ; took seventeen ounces of nourishment, and passed four stools. Phenacetin and caffein āā 1½ grains given at 4 P.M., and at 10 : 45 P.M., when temperature was 104°. Reduced temperature. Enema of normal salt solution given for stimulation of pulse (120–130).

May 19th. — Slept the greater part of night. At 3 A.M. temperature was 99.6°, pulse 104 (no chill). Temperature remained at 101.9° all day. At 7 P.M. rose to 102.8°, pulse 122. Child is improved, much brighter, appetite good (asked for sausage for dinner). Nutritive enema given t. i. d. and retained. Wound dressed. Moderate discharge. Morris drain acted well. Some infiltration anteriorly, under part of incision which was sutured. Packing placed underneath for drain.

May 20th. — General condition improved. Sleeps well. Appetite improved, is limited to fluid diet. Temperature 100.4°, pulse 96, respiration 24 at 7 : 50 A.M. At 5 P.M., after dressing, temperature was 103°. Phenacetin and caffein reduced it to 101.8°, pulse 122, respiration 26. Very little discharge from abscess cavity. Œdema around eye more marked. Sutures removed. Gauze drain inserted. Irrigated with 1 % carbolic. Wet dressing.

May 21st. — General condition improved. Œdema and infiltration of the face lessened. Lowest temperature 101°, pulse 104, respiration 24. Highest temperature 102.8°, pulse 112, respiration 24. Granulations formed in abscess cavity.

May 22d. — Improvement gradually progressing. Sleeps most of night. Bright mentally. Œdema of eye and face much less. Wound dressed as usual. Very slight discharge of pus from cavity. Highest temperature 101.8° (after dressing), pulse 106, respirations 24.

May 23d. — Condition about the same. Slight discharge of pus from cavity. Takes fair amount of liquid diet. Bowels moved. Sleeps well. Highest temperature 102°, pulse 120, respirations 24 ; lowest 100.6°, pulse rate 98.

May 24th. — Usual condition. Temperature in morning 99.2°, pulse 99, respiration 24. At 8 P.M. suddenly rose to 103.2°, pulse 120, respirations 24. Bowels had not moved all day until at about 7:30, when had large stool. No change in mental condition.

May 25th. — Temperature 99.6° A.M.–102.4° P.M. Pulse 98–106. Nux vomica prescribed t. i. d. Some discharge of thick pus from cavity. Œdema of face gone.

May 26th. — In afternoon temperature rose to 103.6°. Phenacetin and caffein given. R. Morris drain left out, and small packing of gauze inserted into abscess cavity, which is almost completely filled. Infiltration about rest of wound almost entirely gone.

May 28th. — Condition generally seems somewhat improved. On May 27th, small collection of pus about the nail of index finger evacuated and wet dressing applied. To-day there was still some pus about nail.

Finger introduced into brain cavity. Some pus and necrotic material came away. It also began to bleed, showing that granulations have formed. Still has afternoon temperature.

May 31st. — Temperature 99–100.6°. General condition good. Very bright mentally. No discharge from cavity, which is much diminished in size. Granulations removed from edge of wound. Sores on back of head. Doing well.

June 5th. — Patient doing very well. Brain cavity filled. For past two days unexplained discharge from middle ear. Temperature normal.

June 8th. — General condition much improved. Boy sits up in bed, and plays with toys. Wound clean and filling up rapidly. Discharge from ear is lessening. Sores on back of head much improved. Sloughs almost entirely freed. Wet dressing applied to them. Temperature 99°–100°.

June 10th. — Yesterday granulations at edge of wound scraped. To-day wound looks well. It is fast closing. Discharge from ear very slight. Temperature normal.

June 12th. — Until to-day temperature has been normal with exception of some afternoons, when it rose to 100°–100.6°. Child seems perfectly well, but, to-day, temperature has been between 101.2° and 102.6°, pulse 98–120, respirations 24–32. Ear examined and nothing abnormal found. No retention, and wound looks healthy. Sores on back of head almost healed. Throat examined, negative. Child does not complain of anything, looks well, has good appetite, and bowels have been freely moved. Sleeps well.

June 13th. — Temperature 101.4°–102.4°, pulse 96–104, respirations 24. Child does not seem at all sick. To-day a moderate discharge has made its appearance from ear of operated side.

June 20th.— Since June 13th temperature has never risen above 100.4°, and to-day it is normal. Discharge from ear almost entirely gone, and deep wound behind closing rapidly. Scalp sores healed.

June 22d.— To-day at 7 A.M. temperature 99°, pulse 104, respirations 24. Child did not feel well during the forenoon. Refused nourishment, complained of headache, and lay in bed instead of going into yard as was his wont. About 11:30 A.M. fell asleep, and remained so until about 1:30 P.M., when he awoke, headache gone, much brighter, and almost as well as usual. His breath had at this time a distinct sour odor. Bowels had moved twice yesterday, but so far not to-day. At 1 P.M. temperature 100.4°, pulse 108, respirations 24. Enema given at 3 P.M., but was ineffectual. About 6 P.M. the nurse girl noticed the child appeared dazed when he awoke, and tried to speak to her, but could not, making signs with his hands. He shortly fell into a deep sleep. At 7 P.M. was awake. About 7:15 child's condition changed suddenly and perceptibly for the worse, became unconscious, eyes wide open, pupils dilated, face very pale, voided urine involuntarily. This state lasted for a few minutes, and then the face commenced to twitch actively, especially the right side. Then there were some slight convulsive movements of the limbs, the pulse was 112, respiration 40. After this, child again dozed off, and at about 8 P.M. was conscious, at first saying only a few words to his father, and then resuming more or less his natural condition, though remaining still somewhat drowsy. About 9 P.M. Dr. Gruening examined the wound, but found nothing abnormal. It was, however, decided, that in the light of the symptoms aspiration of the brain to ascertain whether or not there was retention was justifiable.

Aspiration by Dr. Gruening under ether anæsthesia.

Wound thoroughly cleansed with alcohol. Aspirating needle then passed into brain a distance of one and one half inches through old opening. Passed in three directions, each time with negative result.

June 23d.— About 12 P.M. child recovered from anæsthesia, and insisted upon his father sending for nurse girl. Slept a few hours, and this morning is perfectly normal mentally, though not looking quite as well as on previous days. At 3 A.M., temperature 100°; at 6.30 A.M., temperature 100°, pulse 108, respirations 24. The remainder of the day it remained temperature 99°, pulse 102, respirations 24.

June 24th.— Temperature normal, pulse 104, respirations 22. General condition fine. 1½ grains of calomel had been given on previous day. To-day enema given with very effectual result. Child's every appearance well and healthy. Wound fine and almost completely filled with granulation tissue. External auditory canal has tendency to be closed by granulation tissue.

June 25th.— Discharged. Cured.

A CONTRIBUTION TO THE STATISTICS OF THE EXCISION OF THE SHOULDER-BLADE.

By ARPAD G. GERSTER, M.D.,

Surgeon to Mt. Sinai Hospital; Consulting Surgeon to the German Hospital, New York.

THE removal of the scapula is practised for three causes: *first* and most frequently for neoplasms; *secondly* for chronic suppurative processes, starting either in the shape of an acute infectious osteomyelitis, or produced by a breakdown of a tuberculous, or syphilitic, or an actinomycotic focus; and *thirdly* for extensive compound injuries.

The operation is a rare one. Stephen Rogers published in 1868 the first statistical collection of the then recorded cases in the 56th volume of the *American Journal of the Medical Sciences*, contending that the total removal of the scapula yielded better results than the partial. Ten years later v. Adelmann [1] had collected only 61 cases. Of this number 27 patients died and 34 recovered after the operation. This high mortality is to be attributed to the imperfect hemostasis of the earlier days of surgery, and to the almost unavoidable wound-infection prevailing before the introduction of modern antisepsis. v. Langenbeck, who first demonstrated the possibility and utility of preserving the upper extremity, robbed the operation of much of its deterrent character. But the principal stride forward in attracting the surgeon's favor to this operation was undoubtedly made by the remarkable diminution in the death-rate, directly consequent upon the introduction of the Listerian principle.

We owe the latest collection to the industry of Dr. Wilhelm Schultz,[2] who had, up to 1896, computed the histories of 204 cases, and has made his publication valuable by grouping the material in two divisions. One contained the cases published *before*, the other those that appeared in print *after*, 1875, this date being assumed to be

[1] Zur Geschichte und Statistik der totalen Entfernung der Schulterblätter." *Verhandlungen der deutschen Gesellschaft für Chirurgie*, VII. Congress, Berlin, 1878, pp. 137–147.

[2] *Deutsche Zeitschrift für Chirurgie.* Band xliii., p. 443.

I.

REMOVAL OF SCAPULA, ARM PRESERVED; 80 CASES.

For Tumors, 51 Cases.				For Inflammatory Processes, 12 Cases.				For Injuries, 17 Cases.			
Before 23 Cases	1875	After 28 Cases.		Before 8 Cases	1875	After 4 Cases		Before 17 Cases.	1875	After *None.*	
Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.
30.43%	Due to operation, 17.39 %	64.29 % of which 10.71 % are ascertainedly permanent cures.	Due to operation, 7.14 %	87.50%	12.50%	100 %	———	41.18%	52.94%	None on record.	
	Due to relapse or metastasis, 39.31 %		Due to relapse or metastasis, 17.86 %								
	Other causes not related to operation, 8.70 %		Other causes not related to operation, 7.14 %								
30.43%	66.22 %	64.29 %	32.14 %								
Result unknown, 4.35 %		Result unknown, 3.57 %						Result unknown, 5.88 %			

II.

REMOVAL OF SCAPULA, TOGETHER WITH PART OF CLAVICLE, AND OF THE UPPER EXTREMITY; 102 CASES.

For Tumors, 95 Cases.				For Inflammatory Processes, 7 Cases.				For Injuries, 22 Cases.			
Before 26 Cases	1875	After 69 Cases.		Before 2 Cases	1875	After 5 Cases.		Before 14 Cases	1875	After 8 Cases.	
Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.	Cure.	Death.
34.62 %, of which 30.77 % are ascertainedly permanent cures.	Due to operation, 19.23 %	56.52%	Due to operation, 13.04%	100 %	———	100 %	———	64.29%	35.71%	62.50%	37.50%
	Due to relapse or metastasis, 34.62 %		Due to relapse or metastasis, 27.54%								
34.62 %	53.85 %	56.52 %	40.58 %								
Result unknown, 11.53 %		Result unknown, 2.90 %									

the dividing line between the older or septic, and the newer or antiseptic eras. The literature of the last three years has yielded three more cases, to which I am able to add a fourth new case, bringing the total up to 208 cases, truly a small number compared with almost any other group of standard operations. All of these four patients have recovered. Their small number would not materially change Schultz's statistical conclusions, which, for the sake of greater comprehensiveness, are reproduced here in tabulated form.

The study of these statistical tables will reveal, among others, the following facts:

First, we see that in the history of surgery, up to the year 1875, there were recorded only 90 cases of the removal of the scapula; 42 without, and 48 with, the preservation of the upper extremity. On the other hand, the operation was performed since 1875, that is, in the period of 25 years, 118 times; 82 times without, and 36 times with, the preservation of the upper extremity.

Secondly, the study of the individual histories shows that, among those operated upon since 1875, there is a very large proportion of cases of extensive disease, requiring the ablation, with the scapula, of the upper extremity. Evidently, the improvement in the results of all operative measures, due to antisepsis, served to extend the limits of the indication, and encouraged the surgeon to undertake what would have appeared a "noli me tangere" in olden times.

Thirdly, the removal of the scapula for inflammatory affections, such as caries and necrosis from whatever cause, has not, since 1875, caused a single death, whereas its mortality was 12.50% before that period.

Fourthly, if we compare the histories of cases in which the scapula was removed for injuries, we find that, whereas the scapula alone was removed for that cause before 1875 17 times, this operation was never performed since that date. The high mortality—52.94%—in these 17 cases is mainly to be attributed to wound infection; but was also due to the fact that most of the cases represented gunshot fractures sustained on the battlefields of 1848 and 1864.

Now follow the cases published since the appearance of Schultz's paper.

1. COUSIN. *Resection of the scapula for tuberculous panosteitis.* Boy, 13 years old. The functional result was very good. Abduction to a right angle, carrying forward in the horizontal plane of the arm thus raised, was possible even before the wound was completely closed. *Province médicale*, 1896, No. 39.

2. P. BERGER. *Total resection of scapula and of the external end of the clavicle for a relapsing sarcoma of the right shoulder.* The scapula

and part of the clavicle were removed in a girl 21 years old, for a regional relapse of a sarcoma that had first involved only the suprajacent soft parts. Two months after the operation the patient had a good use of the forearm and of the hand, but abduction and elevation were absent. *Bull. et mém. de la société de chir. de Paris.* T. xxiii. p. 571.

3. M. SCHMIDT. *Total extirpation of scapula for sarcoma.* Man, 57 years old, with a hard tumor in the fossa infraspinata, of the size of a man's fist, of one year's standing. There were a few secondary nodes in the soft parts corresponding to the outer angle of the shoulder-blade. Operation December 5, 1897. Vertical incision along outer margin of scapula, supplemented by a horizontal one carried along spine. Excision of scapula together with all the muscles thereto attached, likewise of the secondary nodes and of several infected axillary glands holding close relations to the axillary vessels. The acromial end of the clavicle was removed, but the head of the humerus preserved. Hemorrhage easily controlled. Healing retarded by marginal necrosis of the skin-flaps and of muscular tissue, but completed in six months. Absence of active movements in the shoulder-joint, but the motility of forearm and hand entirely normal. *Deutsche Zeitschrift für Chirurgie*, Bd. L., p. 394.

4. GERSTER. *Round and spindle-celled sarcoma of the scapula. Extirpation. Cure.* Benjamin L., eight years old, admitted July 1, 1899, to Mount Sinai Hospital. No history of traumatism. Three months before admission, patient complained of pain at the shoulder, where a small swelling was first noticed, that has grown steadily since that time. On admission, the pulse and temperature were normal, the urine normal. The well-grown, sturdy boy's internal organs were all found in a normal state. Over the infraspinatus region of the left scapula, extending almost to the external angle, a hard, elastic, and dome-shaped mass could be seen and felt, firmly attached to the bone, and moving with it, of the size of a small apple. The skin was freely movable over it, and of normal aspect. No dilated veins were present. As an exploratory puncture had yielded a negative result, the differential diagnosis between a tubercular affection and neoplasm had to be cleared up by a probatory incision, which was done on July 5th. The segment of the mass, which was then excised, showed a pale and opaquely transparent, crumbling tissue, which, under the microscope, was recognized to be a rapidly growing round- and spindle-celled sarcoma. The wound was immediately closed by suture.

July 11th.—Chloroform anæsthesia. Two incisions were made, both beginning at the coracoid process, one extending backward and along the axillary border of the scapula, the other along the inner border,

until, enclosing the skin investing the tumor, both incisions converged and met at the inferior angle of the scapula. The supra- and infra-spinatus, and the subscapularis muscles were removed, together with the bone, in one mass. The dissection was begun below, by liberating the angle of the scapula from the margin of the latissimus; then the rhomboidei were severed at their attachments, and likewise the serratus anticus major and teres major. Then came the levator scapulæ, the trapezius, the deltoid, and finally the humeral ends of the teres minor, supra- and infra-spinati, and of the subscapularis. Thus the capsule of the shoulder-joint was exposed and cut away close to the margin of the glenoid fossa, and the tip of the coracoid process being divided with a bone forceps, the whole scapula was removed. Presumably on account of the rapid growth of the tumor, the divided tissues were very vascular, and a large number of ligatures were applied, step by step, so that the loss of blood was not considerable. The axillary space did not contain any infected glands. The large defect in the capsule was closed by a running suture as far as the material permitted, then the stumps of the divided muscles were brought together by several layers of buried sutures, supplying needed covering for the head of the humerus. The skin was also closed by a number of button and catgut sutures, and drainage was secured by a gauze wick extending to the joint. A compressive gauze dressing and a Velpeau bandage, made of starched material, completed the procedure. The patient had borne the operation well, and left the table with a rapid but otherwise good pulse, which, after proper stimulation by enema and the hypodermatic needle, soon came down to nearly the normal standard.

July 12th.—Temperature, 101° F. Pulse, 90; general condition good; tongue moist.

July 16th.—Temperature, 102° F. Change of dressings. A hematoma had formed in the lower recesses of the wound, from which a considerable quantity of sanguinolent serum escaped after the removal of the button sutures. Gauze drainage removed.

July 17th.—Still some fever. The discharge sero-purulent, and drainage imperfect. The upper angle of wound was opened, and a tube was inserted into a recess corresponding to the retracted coracoid head of the biceps.

July 18th.—Counter-incision made, to drain recess below the lower angle of the wound. Tube removed from upper angle.

July 19th.—Pulse and temperature normal. A rapid healing of the large wound followed, and the patient was discharged cured on August 19th.

In December the following conditions were found: A sound and healthy-looking scar; no vestiges of a relapse. The functions of the hand and forearm perfect. A slight abduction of the arm, flexed at elbow, possible to an arc of about 15°. Elevation forward about the same, both due to the clavicular portion of the deltoid. This functional result was uniformly found to be present in all cases where the scapula was removed for a tumor. But it might be much improved, I think, by the bringing about of a union by suture between the scapular attachments of the trapezius and rhomboidei above with those of the deltoid below. Where the scapula was removed subperiostically, for necrosis, the function was, for obvious reasons, infinitely better.

Addendum.

While going to press, my attention was called to an article entitled, "Zur totalen Entfernung des Knöchernen Schultergürtels," by KÖNITZER, in the *Deutsche Zeitschrift für Chirurgie*, vol. lii., p. 594, in which the author computes the results of twenty-nine cases of extirpation of the scapula published since 1896. Of these, in twenty-four cases the operation was done for neoplasms. In twenty-three cases the patients recovered; in one case death followed the operation. Among those that had recovered, a relapse was noted in thirty per cent. of the cases; a relapse was absent one year after the operation in twenty-six per cent. of those that had recovered, and within more than one year's time in twenty-one per cent. In the remaining twenty-four per cent., the results could not be ascertained by the author. By adding this new material to the collection of Schultz, the statistical conclusions will be considerably improved in favor of the operation.

A. G. G.

ACUTE DILATATION OF THE HEART IN INFLUENZA OF CHILDREN.

F. FORCHHEIMER, M.D.

ACUTE dilatation of the heart has been observed in a number of infectious diseases either as the result of direct or indirect affection of the heart.

As direct affection of the heart may be considered those forms in which dilatation, and as a result myocardial insufficiency, is produced by primary disease or abnormal functional activity of the myocardium itself.

As indirect affection those forms may be considered in which the dilatation is the result of functional or diseased conditions of all those various apparatuses which in one way or the other have a direct influence upon the heart's activity, — the respiratory apparatus, the nervous system, the blood-vessels, the kidneys.

Under the first heading, then, we can take into consideration diseased conditions of the myocardium, its muscular or connective-tissue substance; and therefore, to a greater or less extent, the intracardiac nerve supply.

Under the second heading must be included the consideration especially of those conditions tending directly to change the intracardiac pressure as the result of increased resistance to the outflow of blood from the heart; and the changes that take place in the cardiac centres, and the extra-cardiac nerves.

When the dilatation of the heart goes beyond certain limits, then we have produced symptoms of myocardial insufficiency. The appearance of these symptoms varies in individuals; and it is difficult in an individual case to state when the line of demarcation, as between physiological and pathological dilatation, has been passed. It has been my observation that children in most instances will recover from that which seems a pathological dilatation with incredible rapidity, in that respect differing greatly from the adult; again, this is largely a question of individuality, as we find some children who not only

seem predisposed to this condition, but in whom not only all the symptoms of myocardial insufficiency develop as completely as in the adult, but also continue to do so upon comparatively slight provocation. While this is the case, yet the prognosis differs in the two periods of life, in that, once the great storm of symptoms has passed over, the child's heart recovers itself more quickly, and develops into what seems a normal heart; at least, without compensatory hypertrophy, while in the adult compensatory hypertrophy is the rule.

The clinical picture produced by acute dilatation of the heart in influenza has been known to nearly all the authors who have written upon the subject of influenza during the last twenty years; but it was left to Huchard (1890) to give a special name to this condition,—"la grippe cardiaque." For myself, I have always believed that terms like renal typhoid, or cardiac grippe, are of no especial benefit to us, in that, after all, in these conditions it is only a local manifestation of a general affection that we are dealing with; a fact that should be thoroughly appreciated and fully recognized at all times. For this reason, I have preferred the title, "Acute Dilatation of the Heart," as it at least expresses the definite condition which is always present; one that we find in septicæmias of all varieties, in the acute exanthemata, in whooping-cough, diphtheria, influenza—in short in nearly all the acute infectious diseases, either as the result of the specific, primary infection, or as the result of the secondary infections which we are so apt to find in all of these conditions. To the great Dublin clinician, Graves, is probably due the credit of having made a distinct clinical picture of the characteristic conditions, and which I am about to consider (*Clinical Lectures on the Practice of Medicine*, 1843, "Influenza)." He says: "One of the most singular features of influenza is the extraordinary degree of dyspnœa witnessed. . . . and even in many cases when the bronchial mucous membrane is but slightly engaged, the amount of dyspnœa is remarkably great . . . in many cases it is intermittent, or at least undergoes remarkable exacerbations and remissions at certain hours of the day and night." . . . "It would appear that the respiratory derangement depends on the same general cause which produces the whole line of symptoms; and that it might exist even when there was no bronchial inflammation at all." This same condition is referred to with more or less detail by the German authors Biermer, Zülzer, and Kormann; but it is to West (*Lectures on the Diseases of Infancy and Childhood*, Phila., 1874., p. 281) that we owe a description of a more intense development of this train of symptoms. In the epidemic of 1856 West observed a combination of symptoms of which dyspnœa was the principal one, disappearing

in two or three days, followed by "extreme depression, cool, moist skin, a very feeble pulse, and labored respiration. . . . In this condition the children, though quite conscious when roused, lay generally dozing, while, though the somewhat livid hue of the lips and surface seemed to imply the existence of some serious mischief in the lungs, there was nothing to be heard but a large moist râle." He concludes by stating that "on several occasions," under increased depression, a fatal issue followed.

In this country we are now in the tenth year of successive epidemics of influenza; and the opportunity for study of its various manifestations and complications, both in private as well as in hospital practice, has been exceptionally favorable on account of the number of cases and the differing course of the disease in different epidemics. As a result, many of us have seen the conditions above described not infrequently. An infant or child is taken down, in the course of a house epidemic of influenza, with high fever accompanied by symptoms on the part of the respiratory or alimentary tract, possibly with a catarrhal or follicular angina tonsillarum, and within 12–24 hours there develops dyspnœa—or better, tachypnœa, for it is perhaps more correct to refer to the condition of rapid breathing in this form of the affection as tachypnœa, as all other evidences of dyspnœa are wanting, such as cyanosis and movements of the accessory respiratory muscles. The child lies in bed with congested skin, sleeps a great deal, yet is easily aroused; the tachypnœa is represented by rapid breathing (60 to 70 or more per minute), without disturbance of the normal relation between inspiration and expiration, therefore, neither inspiratory nor expiratory dyspnœa. In some cases the breathing is not very much more rapid than the normal. The pulse is very rapid, depending upon the age of the patient, frequently intermittent; but otherwise normal. Physical examination of the chest reveals nothing, or possibly evidences of a bronchitis in the large tubes; the heart dulness is always found broadened, with no bruits as a symptom of this affection, although in one instance I found an endocarditis developing after the acute dilatation had run its course. The urine, which was examined in every instance, was invariably found to contain no albumen. In the course of from 24 to 72 hours all these symptoms disappear, the temperature, pulse, heart, and respiration become normal, the whole condition of the child changes, it returns to its normal state, and then remains well; or the various symptoms of a more prolonged attack of influenza develop. I have never seen this mild form develop into the more severe one, as seen by West; all my severe cases have developed in a different way, as will be seen presently.

As to the etiology of this condition various and varying views have

been expressed. Graves believes that the poison of influenza acts "on the nervous system in general, and on the pulmonary nerves in particular," and that the condition produced is one to be likened to the one following section of "the eighth pair of nerves" in animals. Biermer (*Virchow's Handbuch*, V., vol. vi.) thinks that the symptoms are due to a pulmonary congestion; Zülzer (*Ziemssen's Handbuch*, II., Bd. iii.) looks upon the condition as one akin to nervous asthma, more like a motor neurosis of the diaphragm and the bronchial muscles; Sansom (*Medico-Chirurgical Transactions*, 1894, p. 290) says that "the cardiac crises during the acute stage of the disease might be due to involvements of the nerve elements in the bulb, the remote symptoms should be ascribed to a neuritis affecting the sympathetic ganglia, the vagus, and the sensorimotor nerve trunks."

Against the acceptance of congestion of the lungs as the cause of these symptoms speaks the fact that there are no physical evidences in the lungs which show the existence of this rather indefinite condition. These might have eluded detection on the part of one or more observers, but all those who have seen these symptoms remark upon the disproportion between physical signs in the lungs and the rapidity of breathing. It seems more satisfactory, therefore, to accept a nervous origin for these symptoms, although there is no direct evidence in favor of this view. Merklen and others, it is true, have found lesions in the vagus in cases of tachycardia; but we have here a condition producing especially tachypnœa. Myocarditis can be excluded because of the transitory nature of the affection, although in the grave forms of dilatation this has been observed (Huchard). Neither does it seem possible that the cardiac branches alone are affected, otherwise the tachycardia would be the prominent symptom, while this is only relative, and the tachypnœa the dominant symptom. It is, therefore, more reasonable to suppose with Sansom that there is some affection of the bulb affecting both the respiratory and the cardiac accelerating centres. The source of the irritation would be a toxin, hypothetical as far as the influenza bacillus is concerned, but positive as to some of the pus producers which habitually accompany influenza. It would be fully in accord with the nervous origin of these symptoms that we find broadening of the heart's dulness. The production of acute dilatation of the heart, as the result of disturbance in innervation, has been conclusively proven in many diseases. The second class of cases to be described demonstrates this more fully.

The grave form of the affection presents an entirely different clinical picture. Here there can be no question as to the origin of symptoms,

at least, as all the manifestations can be satisfactorily explained by considering the heart. Huchard "Sur quelques formes cliniques de la grippe infectieuse," *Bulletins et Mémoires de la Société médicale des Hôpitaux de Paris*, S. 3, vol. vii., p. 93, 1890) gives the following concise description of this condition (La Grippe Cardiaque): "The attack manifests itself by syncope and faintness, which may become fatal, by slow pulse, by arrhythmia, or intermittence, by grave symptoms of cardiac collapse; and, sometimes, by pains resembling angina pectoris." One of his patients had a very rapid pulse, with a feeling of suffocation upon making the slightest movement. How this form of acute dilatation of the heart presents itself in children will be best shown by the following histories of two of the four cases I have seen:

The first was in a girl twelve years of age, weighing about 130 pounds, who previously had had rickets, diphtheria, typhoid fever, pertussis, and measles, from all of which she had recovered without any sequelæ. The family history is good. The child was taken sick with fever and sore throat on March 6, 1897, from which she recovered in a few days, and this attack left her with a cough which continued until I was called in to see her again, on June 5, 1897. This cough, which I did not hear before the latter date, but heard too often afterward, was the cough that we so frequently hear in influenza, and which has, not inaptly, been called pertussoid. On the morning of June 5, 1897, the child had fever (101°), and the cough, as far as number and intensity of paroxysms was concerned, had grown very much worse. In the evening the temperature had risen to 105°, with pulse and respiration in proportion; the cough was now so bad that with each paroxysm the child complained of pains about the lower region of the thorax and in the abdomen. Physical examination revealed the evidences of a bronchitis, large moist and dry râles, and a large superficial heart dulness almost to the right border of the sternum and to the mammary line to the left, the latter to be explained, possibly, by the obesity of the child. No evidences of consolidation in the lungs, nor were there any abnormal ausculatory phenomena to be found in the heart. The next few days passed in the same way, the temperature, pulse, and respiration keeping up, the cough becoming worse, and vomiting taking place with nearly every paroxysm of coughing. As a result of this constant coughing, the abdominal pains became so great, constipation and vomiting being present, that a suspicion of abdominal inflammation arose, which, however, was soon dispelled; in other words, this child was now suffering from recrudescence of the influenza. On the morning of June 9th I was suddenly called to see the child, and found her in the following condition: she was in a state of collapse; she was lying in bed in a soporific condition, from which she was easily aroused; the skin moist, slightly cyanosed, the face swollen, breathing at the rate of 65 per minute without any effort apparently; the pulse had come down from 120 beats to 70 per minute, and was weak and intermittent. The slightest effort on her part, such as turning in bed, caused the frequency of respiration to be increased and the pulse to become more irregular and weaker. After an attack of coughing the pulse became almost imperceptible, and the breathing ran up to as high as 70 per minute. A thorough physical examination was not to be thought of on account of this condition; but auscultation of the heart showed nothing abnormal; while percussion, even in the recumbent position, showed the right border of the heart about one half a centimeter to the right of the sternum. After two days the condition changed somewhat; large doses of codeia were added to the medication, as it seemed imperative to control the paroxysms of cough as much as possible, on account of their untoward effects upon the heart. Now the pulse gradually began to increase in frequency, going up to 140 beats per minute; the dyspnœa was not so constant, there being a difference of between 15 and 20 respirations during the condition of

being awake or asleep; but motion, swallowing, or any excitement would make this difference disappear, and an attack of coughing would raise the number of respirations even higher than it was before (as high as 90 per minute). The pulse continued rapid all this time, never going below 120 beats per minute, and continued intermittent, although not quite so weak as on the previous days.

Physical examination of the lungs did not reveal anything more than at first, but the heart showed remarkable changes. The superficial dulness was increased to 1 cm. to the right of the sternum; and after attacks of coughing to as much as 2 cm., depending upon the intensity of the paroxysms; no appreciable change to the left. Over the tricuspid area, as well as at the apex, was heard a soft systolic bruit, which also was increased in intensity after paroxysms of coughing. Bruits were also heard at the base, especially after the paroxysms of coughing, but they were more difficult to localize, and were conducted in such a way that it was impossible to ascribe them to one valve or another. Gradually, as the result of treatment, all these symptoms began to disappear; the pulse came down from this time until September, 1897, so that its minimum was 87, and its maximum 120 beats per minute; the latter upon exertion. As soon as it was possible graded exercises were used, and the patient was discharged as well the middle of December, having been ill for five months, with diurnal changes for better or worse, but on the whole with a constant tendency toward improvement. On May 7, 1899, I examined the patient for the last time. I then found her, having lost her fat, having grown four inches, and with a normal heart which was no longer insufficient under any conditions of exercise or fatigue.

One other case did not materially differ from the preceding except that the manifestations were not so severe and did not last so long. In addition, this last child was younger and not so fat, so that the contributing cause of obesity of the heart was absent in her. In the former case there were some peculiarities that must be taken into consideration. The child was unusually fat for her age; it is, therefore, not unlikely that there was some obesity of the heart, rendering the myocardium less resistant to strain than it would have otherwise been. Three months of continued coughing certainly did not add to the strength of the myocardium, so that when a renewed invasion of influenza occurred cardiac collapse ensued, which first manifested itself by great asystole, and afterwards by a decided and distinct acute dilatation of the right heart, with relative tricuspid insufficiency.

It is difficult to decide upon the real condition which first caused this cardiac collapse; but it was my distinct impression at the time of first seeing this patient that I had to deal with an uncomplicated case of myocardiac insufficiency, the result of heart strain, the factors producing the heart strain being the obesity and the whooping-cough-like paroxysms of coughing. It is outside of the province of this paper to discuss the rôle played by the influenza in producing the pertussoid; the mechanism of production of heart dilatation in this case was the same as that found in whooping-cough, to which so many authors have called attention. That the toxin had something to do with the improper innervation of the heart it is impossible to deny, but just as impossible to affirm.

The following case again illustrates peculiarities, but seems to prove especially that the myocardium is the part of the heart that is affected. The patient, a little boy two years and three months old, of good antecedents, artificially fed as an infant, had had influenza when four months old, followed by acute otitis media; in October, 1897, when fifteen months old, he had another attack of influenza, which was followed by the peculiar and irregular fever so common after this disease, which lasted for two months, and which resulted in great depression, anæmia, and loss of flesh (*no malaria*, *no typhoid*). In June, 1898, he contracted whooping-cough, which ran its course in a very mild manner (maximum, ten paroxysms in twenty-four hours).

In October, 1898, he had another attack of influenza; as the little fellow had three attacks of influenza, the fact must be emphasized that, in every instance, all the members of the family were affected with one or the other form of the disease, so that the diagnosis was perfectly clear. In this last attack the pertussoid nature of the cough was clearly demonstrable, as would have been the case, in all possibility, with any cough which would have developed so shortly after an attack of whooping-cough. Early one morning after an unusually severe attack of coughing, "he turned blue in the face, and fell fainting in my arms," as the mother expressed it. Two hours after this I saw him, and found him cyanosed, sweating, breathing very rapidly, and with a rapid, irregular, weak pulse. Physical examination of the lungs showed the evidences of bronchitis. Inspection of the cardiac region showed pulsation over the whole area; in addition there was epigastric pulsation. The heart impulse was diffuse, felt most distinctly in the fourth interspace to the left of the mammary line; percussion verified this as the left border of the heart. Toward the right the dulness extended to one half cm. beyond the right border of the sternum, and above began at the fourth rib. There was no bruit. The tachypnœa was influenced by the slightest exertion, but the heart did not increase materially in size. The patient was kept in bed and under treatment for five weeks, and then made what seemed from physical evidence a rapid recovery. During the summer he had another attack of tachypnœa, which, however, lasted only a few days. When brought back to me in the fall there was no evidence of cardiac trouble. The following winter (1898–99) was uneventful; but the mother, preparatory to leaving for the summer, in June, 1899, asked me to examine the child again, which I did, and found the heart normal. The fatigue of a very hot journey and an attack of illness, accompanied by fever and gastro-intestinal symptoms, again brought on the same train of symptoms described above, the latter being diagnosticated by the local

physician as due to heart trouble. This attack lasted for two weeks. My notes made on September 2, 1899, show the following as the condition of the heart: Apex beat immediately below the nipple in the fifth interspace; superficial dulness begins at the apex, the lower border of the fourth rib, and the left border of the sternum. No bruit. Since the last attack the child has been well. The peculiarities of this case are: That the myocardium undoubtedly was weakened by the previous attack of whooping-cough, although no evidences of this weakness could be detected, and that this weakness has continued, so that when sufficient demand is made upon the myocardium, high fever with gastro-intestinal infection, for instance, the same symptoms return. On account of the long periods of rest which the child has enjoyed, it does not seem reasonable to suppose that the nervous system of the heart, except as implied in the myocardial structure, plays any important rôle in the production of these symptoms. Again, at no time during the course of the disease was there bradycardia. It seems that, here again, we are dealing with a purely mechanical dilatation of the heart, due to increased resistance to the outflow of blood from the right side of the heart. The fourth case had no predisposing conditions of myocardium, as had the other three; and developed after the febrile stage when the pertussoid was at its height. Here the symptoms came on just as rapidly; physical examination revealed an enlarged right heart; but the whole condition lasted only ten days, leaving the heart normal. The dominant symptoms in all these cases were on the part of the heart, and the peculiar respiration which I have only seen in two other conditions; in pericardial effusion, and the fugitive œdematous condition of lung that frequently precedes pneumonic consolidation in influenza without cardiac changes. It is this latter observation that has induced me to explain all the cases of the milder form by taking into consideration the effect of a toxin upon the nervous system in producing acute dilatation.

As to the grave forms, observations made on four cases are not sufficient to form a definitive opinion, although in all of them the direct cause of the trouble seemed to be heart strain, in two of them there being a predisposing weakness of the myocardium. It is possible that in those two cases a myocarditis was going on at the same time, although the short duration of the attack in one of the cases, and the complete restitutio ad integrum in all of them, seems to preclude this. Under all circumstances, it seems to me that we are justified in calling this condition acute dilatation of the heart.

All the reflections on the subject of affections of the nervous system of the heart in influenza are purely theoretical; as yet, no changes

have been described in any one of the various parts of the nervous system of the heart, as has been done for scarlatina and diphtheria. Until this is done it seems safer to hold fast to that which we can verify, which has been expressed by Goodhart (*Albutt's System of Medicine*, vol. i., p. 602) in a similar case, as follows: "The only conclusion that seemed possible was that an acute dilatation of the heart had taken place."

According to my own observation we are justified in assuming two forms of heart dilatation in influenza: one presumably produced by the action of the toxin upon the nervous system of the heart, and possibly upon the myocardium; the second form occurring in such conditions in which outflow of the blood is materially interfered with on account of mechanical conditions.

The first form, according to the excellent observations made by West, undoubtedly may end fatally; although this has not been the case in my own experience.

The second form is one that lasts much longer than the first, but in children has a tendency to recovery.

ERFAHRUNGEN UBER CHOREA.

VON PROFESSOR C. GERHARDT.

DIE Kinderheilkunde hat meine langjährigen, freundschaftlichen Beziehungen mit Professor A. Jacobi angebahnt, deren ich heute dankbar gedenke. Er ist der Kinderheilkunde treu geblieben und hat uns immer wieder reiche Ernten seiner Forschung auf diesem Gebiete dargeboten. Ich dagegen bin soweit davon abgedrängt worden, dass es mir heute schwer fällt, ihm noch ein Bündel thatsächlicher Beobachtungen als Festgruss darzubringen, die wenigstens einige Beziehung zur Kinderheilkunde haben.

In den vierzehn Jahren meiner Thätigkeit in Berlin kamen in meine Klinik 55 Choreakranke, über die ich Aufzeichnungen habe. Manche frühere und anderweite Erfahrungen sollen hier nebenbei benützt werden. Zwei Fälle von Chorea gravidarum lasse ich hier bei Seite. Kranke unter 12 Jahren kommen in Behandlung der Kinderabtheilung und sind desshalb hier nicht vertreten. Von meinen 32 weiblichen und 23 männlichen Kranken standen im Alter

von 10–15	Jahren,	30
" 16–20	"	21
" 21–25	"	3
" 26–30	"	1

Fast alle stehen somit im Alter von 10–20 Jahren. *Ursachen: Vererbung* konnte einmal angenommen werden; die Mutter war in der Jugend, ein Bruder ist jetzt noch chorealeidend. *Erbliche Belastung* ist bei sieben Kranken wahrscheinlich. Zweimale hat die Mutter Hemicrania, daran litt einmal auch der Sohn schon vor seiner Chorea; dreimale sind Geschwister epileptisch, je einmal ist die Mutter geisteskrank, der Vater Trinker. In vielen Fällen werden frühere Rachitis oder Scrofulose, Blutarmuth, oder Schwächlichkeit erwähnt. Wie günstige Vorbedingungen für die Entstehung der Krankheit solche Zustände bieten, zeigte ein Vorgang auf der Kinderabtheilung in Würzburg. Ein Kind mit Chorea trat ein. Bald fingen einige scrophulöse und rhachitische Kinder an zu zucken. Erst die Verlegung des

ersten Choreafalles schaffte Ruhe. Da dies sich öfter wiederholte, wurde es schliesslich Regel, choreakranke Kinder gleich in die Säle der Erwachsenen zu verlegen. Jetzt sehe ich manchmal, dass Hysterische durch die Nachbarschaft eines Choreakranken zu ähnlichen Zuckungen angeregt werden. Die Geschichte vieler Chorea-Epidemieen, theils in einigen Gegenden, theils in Spitälern und Erziehungsinstituten, zeigt zur Genüge, dass es sich bei Chorea imitatoria nicht um ein Contagium vivum sondern in der That nur um Nachahmung handelt, allerdings nur bei Personen, die durch Anlage, Kränklichkeit oder abnorme Einflüsse von Lebensweise, körperlicher oder geistiger Diät, besonders dazu geneigt oder vorbereitet sind.

Die Untersuchung des Stuhles auf Eier von Entozoen wurde bei unseren Kranken regelmässig vorgenommen, aber sie hatte dürftige Ergebnisse. Zwei hatten Oxyuren–einer Trichocephalen–Eier. Auch diese Würmchen schienen auf Entstehung und Verlauf der Chorea ohne Einfluss gewesen zu sein.

Während nur bei zweien unserer Kranken *Nachahmung* als Ursache wahrscheinlich war, konnte bei 22 das Vorausgehen schwerer Gemüthsbewegungen festgestellt werden. Beispielsweise waren es viermale Todesfälle, deren Anblick erschütternd wirkte, einmal eine Ohrfeige in der Konfirmandenstunde, einmal Liebeskummer, der zu einem Vergiftungsversuche führte, Fall ins Eiswasser u. s. w. Von diesen 22 Kranken hatte die Hälfte weder zuvor Polyarthritis gehabt, noch auch zur Zeit der Beobachtung Erscheinungen eines Klappenfehlers. Von der Bedeutung der Schulstrafen für die Entstehung der Chorea lieferte früher die Klasse einer Lehrerin in einem Dorfe bei Würzburg eine Reihe von Beispielen in die Klinik. Unter den hiesigen Kranken sind sie ausser jener Konfirmandin nur noch einmal vertreten. In beiden Fällen von Nachahmung war auch kein Klappenfehler vorhanden.

Polyarthritis, hie und da auch Monarthritis rheumatica, meist früher vorausgegangen, einigemale unmittelbar vor Chorea, auch während derselben entstanden, wurde 20-male angegeben. Einmal beherrschte sie das ganze Krankheitsbild, die Zuckungen traten nur zwischendurch auf. Die Herzuntersuchung bei unsern 55 Choreakranken, von denen 20 Polyarthritis gehabt hatten, begründete in 35 Fällen die Annahme eines Klappenfehlers, fast bei allen eines Mitralfehlers. Bei 12 weiteren Kranken bestanden vorübergehend systolische Geräusche am Herzen oder sonstige Erscheinungen, die nicht mit Sicherheit die Annahme eines Klappenfehlers erwiesen ; endlich 8 dieser Kranken hatten reine Herztöne und auch sonst völlig normale Verhältnisse am Herzen. Unter den Kranken, die Polyarthritis gehabt hatten, boten 3 reine Herztöne, 2 zweifelhafte Zeichen am Herzen. Somit hatten 20 Kranke

Herzfehler, bei welchen Polyarthritis nicht vorausgegangen war. Von diesen hatten früher 8 Scarlatina, 7 Diphtherie, einer Influenza gehabt. Nächst Polyarthritis wird Scarlatina als diejenige Grundkrankheit betrachtet werden müssen, die am häufigsten endocarditische Chorea verursacht.

Die Angaben in der Literatur beziehen sich zumeist auf "rheumatische Grundlage" der Chorea. So finde ich in zwei Dissertationen von Arthur Zimmermann, Zürich, 1891, und H. Meyer, Basel, 1894, übereinstimmend angegeben, dass 80 % der Fälle "causal mit Rheumat. acutus in Verbindung zu bringen" waren. Andere Statistiken gehen weit auseinander. Bald liegt allen Fällen, bald nur ganz wenigen Polyarthritis zu Grunde. Ich glaube dass man die Frage nach dem Zusammenhange der Chorea mit Polyarthritis und mit Endocarditis getrennt behandeln muss und dass man nicht von rheumatischer Grundlage, sondern nur von vorausgegangener oder mitbestehender Poly- oder Monarthritis sprechen sollte. Frische Entzündung der linken Vorhosfklappe ist der constanteste Befund tödtlich verlaufener Choreafälle. An dieser anatomischen Thatsache muss man festhalten. Aetiologisch klar sind in gewissem Maasse jene Fälle, in welchen während einer Polyarthritis, oder nachdem eine solche einen Herzfehler hinterlassen hatte, oder bei anderweit herzkrank gewordenen Personen Chorea von selbst oder unter begünstigenden äusseren Umständen entsteht. Hier hat man das volle Recht von Chorea endocarditica (rheumatica) zu sprechen. In einer anderen Gruppe von Fällen ruft bei einem seither anscheinend gesunden Kinde, das höchstens vor ein paar Jahren Scharlach oder Diphtherie gehabt hatte, Schreck oder drgl. Veitstanz hervor. Während dieser Krankheit hört man andauernd oder zeitweise ein systolisches Geräusch an der Herzspitze, vielleicht auch den zweiten Pulmonalton gespalten oder verstärkt. Nach Ablauf der Krankheit hören auch die krankhaften Erscheinungen am Herzen wieder auf. Hier kann man wenigstens mit einiger Wahrscheinlichkeit annehmen, dass es sich gleichfalls um endocarditische Chorea gehandelt habe.

Bei einer dritten Gruppe von Fällen entsteht Chorea durch Gemüthsbewegungen. Die Herztöne sind während der ganzen Krankheit und vorher und nachher rein. Muss man in diesen nicht ganz seltenen Fällen der Theorie zu liebe latente Endocarditis annehmen? Entsteht diese durch die Chorea oder musste die latente Endocarditis schon da sein, damit Schreck oder drgl. die Chorea auslösen konnte? Wenigstens die durch Nachahmung entstandene Chorea und die ganzen Epidemieen von Chorea wird man kaum mit Endocarditis in Beziehung bringen können. Eine einheitliche Choreaursache lässt

sich vorläufig nicht begründen; man wird zunächst auf scharfes Beobachten und auf Sammeln vieler Thatsachen angewiesen sein.

Etwa ein Viertheil, dreizehn, unserer Kranken, hatten früher schon Chorea gehabt; 6 einmal, 3 zweimal, je einer 3-, 4-, 5-, 6-male. War anfangs nur eine Seite betroffen, so konnte dies bei den Rückfällen wieder so sein, oder es konnten auch bei dem Rückfalle die Zuckungen doppelseitig auftreten. Für einseitigen Beginn des Leidens findet man hie und da ursächliche Anhaltspunkte in der Beschäftigung der Kranken oder in äusseren Einflüssen. Eine junge Dame bekam als Braut Chorea, während sie fleissig an ihrer Ausstattung an ihrer Maschine nähte. Die Zuckungen begannen bei ihr an einem Finger der linken Hand, der besonders stark thätig sein musste, und waren vorwiegend auf die linke Körperhälfte beschränkt. Von meinen klinischen Fällen erwähne ich: eine Kranke musste seit ½ Jahre in einer Fabrik täglich 11 Stunden Glimmer mit der Scheere schneiden; ihre Zuckungen begannen an der rechten Hand. Ein Steinmetzlehrling, 15 Jahre alt, bekommt zuerst beim Steinezuhauen Zuckungen im rechten Arm, dann auf der ganzen rechten Seite, später auch auf der linken. Ein Handschuhmacher, der selbst an linksseitiger Chorea litt, erzählt, das in demselben Geschäft drei andere Gehülfen alle mit rechtsseitiger Chorea erkrankt seien. Einer 15-jährigen Posamentierarbeiterin fiel eine eiserne Waschschüssel auf die rechte Hand; sie erschrak heftig, konnte am nächsten Morgen Nichts mehr in der rechten Hand halten; nach einigen Tagen verbreitete Zuckung. Diese Krankheitsanfänge an überangestrengten oder schmerzhaft verletzten Gliedmassen erinnern an gewisse Erfahrungen des Beginnens polyarthritischer Erkrankungen an ähnlich geschädigten Gelenken.

Krankheitserscheinungen.—Wir sahen in Fällen *schwerer Chorea* ausser stärkeren geistigen Störungen Aphasie dreimale, unvollständige Sprachstörungen siebenmale, epileptische Anfälle zweimale, unwillkührliche Entleerung von Stuhl und Urin zweimale, ebenso Beginn mit einer Ohnmacht zweimale, einmal Schluchzen. Die schwere Erkrankungsform hat ausserdem unordentliches Aussehen des Kranken, Heftigkeit der Zuckungen bis zu Selbstverletzungen und Nothwendigkeit von Zwang und Schutzmassregeln, erschwertes Einschlafen zu Kennzeichen. Von der mannichfaltigen Gestaltung der Symtome sei nur erwähnt, dass sie manchmal an die Ursache des Ausbruches der Krankheit erinnern. So sah ich einmal bei einem Knaben, der unversehens in der Badeanstallt ins Wasser gestossen worden war, in der alsbald ausgebrochenen Chorea eine Art von Schwimmbewegungen vorwiegen. Er bekam später noch epileptische Anfälle und starb nach einigen Monaten. Die Patellarrflexe sind mitunter schwach, anderemale stark, bei

einigen Kranken mit Hemichorea auf der kranken Seite gesteigert, aber nicht bei allen. Die Körperwärme war nieder, nämlich wenn man von fortbestehender oder wiederkehrender Polyarthritis, von arsenikalem Fieber und anderen besonderen Fieberursachen absieht, meistens zwischen 36° und 37° Celsius. Die Athmungszahl war meist nicht oder wenig beschleunigt, wenn auch die Reihenfolge der Athmungen in schweren Fällen unregelmässig war. Der Puls war nur bei beträchtlichen Herzfehlern einigemale unregelmässig; dagegen zeigte die Pulszahl selbst in Zeiten niederer Körperwärme fast auf allen Curven einzelne auffällige Sprünge, so dass sich unter 55 Curven 45-male Gipfel von mehr als 100 Schlägen vorfinden, während Temperaturen über 37.8 nur in 11 Fällen verzeichnet sind. Noch auffälliger schien das Verhalten des *Urines*. Voraus sei bemerkt, dass Albumen im Harne bei täglicher Untersuchung sich nur dreimal fand, zweimale erklärlich durch Nephritis aus anderen Ursachen, einmal nur spurweise und vorübergehend. Bei der Unwahrscheinlichkeit täglich die genaue Gesammtmenge zu erhalten, wurde nur das spezifische Gewicht der in 24 Stunden gesammelten Menge bei nachstehenden Vergleichen in Betracht gezogen. Dieses zeigte fast bei allen, auch bei ganz fieberlosen Kranken, derartige Schwankungen, dass die beiden letzten Zahlen desselben zeitweise, anscheinend regellos, auf das Doppelte stiegen. So ergab sich bei einzelnen Kranken: 1008 und 1018, 1007 und 1030, 1004 und 1036, u. s. w. Schon in älteren Arbeiten, so bei Todd, Beale, Handfield Jones finden sich derartige Angaben. Driso (Würzburger Dissertation) erwähnt einmal 1030 sp. Gew. Auch in der erwähnten Züricher Dissertation von Arthur Zimmermann finden sich bestätigende Aufzeichnungen. So schwankt in seinem Falle, 2 das spez. Gewicht zwischen 1014 und 1025, Fall 5 = 1010 und 1028, Fall 10 = 1012 und 1025. Weitere Einsicht in diese Verhältnisse gewähren die von Zimmermann beigebrachten Zahlen über die täglichen Harnstoffmengen. Diese betrugen in seinem Falle 12 = 7 — 23 gm., Fall 13 = 21 — 61 gm. bei einem 11-jährigen Mädchen, Fall 21 = 21 — 16 gm., Fall 22 = 7 — 31 gm. Man muss darnach annehmen, dass in vielen Chorea-Fällen trotz niederer Körperwärme an einzelnen Tagen Stoffwechselstörungen stattfinden, welche jenen des Fiebers ähnlich sind. Trotz dieser Störungen nahm das Körpergewicht bei allen unseren Kranken zu während der Behandlung, nur mit sechs Ausnahmen.

Ausgänge.—Das Endergebniss war in Kürze folgendes: Gestorben 1, noch in Behandlung 1, Ausgang unbestimmt 2, ungeheilt auf Verlangen entlassen 5, gebessert auf Verlangen entlassen 11, geheilt 35. Manche derer, die gebessert oder ungeheilt austraten, waren schon zuvor in anderen Hospitälern mit gleichem Erfolge behandelt worden.

Von den Geheilten standen 26 zwischen 30 und 70 Tage in Behandlung; die kürzeste Dauer bis zur Heilung war 9, die längste 180 Tage.

Behandlung.—Zumeist wurde Arsen gegeben, dabei oft Fieber und Herpes beobachtet; wenn dieses Mittel versagte, kam oft Campherbrom in Anwendung; bei Schlaflosigkeit bewährte sich Choralhydrat. Auf sorgfältige körperliche Pflege, reichliche Ernährung und mässige Anwendung kalter Abreibungen wurde besonderen Werth gelegt. Man suchte auch in schweren Chorea-Fällen Reinlichkeit und ordentliches Aussehen aufrecht zu erhalten. Andauerndes Bettliegen wurde womöglich vermieden. Sobald thunlich wurden den Kranken leichtere Beschäftigungen aufgetragen.

THE CARDIO-VASCULAR CHANGES IN BRIGHT'S DISEASE.

By JAMES TYSON, M.D.

BY cardio-vascular changes in Bright's disease I mean those changes in the heart and blood-vessels which are more or less characteristic of the disease. The characteristic alterations in the heart are mainly of the muscular substance and primarily of the nature of hypertrophy. For although valvular changes are also at times found associated with renal disease, they are not commonly a consequence but either a cause, coincidence or possibly a result of the same cause. Far more frequently any valvular disease of the heart which is associated with renal disease precedes the latter and is its cause, passive congestion being the most frequent result. To the muscular change alluded to may be added a mild degree of interstitial myocarditis resulting in a corresponding increase of intermuscular connective tissue.

The hypertrophy alluded to was indeed noted by Bright as early as 1827. The clinical evidence of its presence is found chiefly in the displaced apex beat and accentuated aortic second sound, though an enlargement of the percussion area may also be demonstrated. It may invade both ventricles or the left ventricle alone, never the right ventricle only. According to Bamberger's statistics, of the few hypertrophies associated with acute cases 54 % were of the left ventricle, of those associated with chronic cases 53.4 % were of the left ventricle, and of atrophic cases 5 2.5 % were of the left ventricle. Thus it will be seen that the hypertrophy was confined to the left ventricle in surprisingly near one half of all cases: both ventricles being invaded in the remainder, or a little less than half, while hypertrophy was in no instance confined to the right ventricle. Senator[1] claims that simple hypertrophy is more frequently associated with genuine contracted kidney, while eccentric hypertrophy or hypertrophy with enlargement of the corresponding cavity is more common in other forms, including the arterio-sclerotic variety of contracted kidney and parenchymatous nephritis.

[1] Senator, *Die Erkrankungen der Nieren*, Wien, 1896, S. 88.

The anatomical features of the hypertrophic change being settled as nearly as may be, the consideration of their constancy in association with renal disease naturally follows. First, while it cannot be denied that hypertrophy occasionally develops rapidly in acute nephritis, it is mainly found associated with chronic renal disease, and for the most part in some one of the forms of contracted kidney. On the other hand, while it cannot be said that contracted kidney never exists without cardiac hypertrophy, instances are so few that hypertrophy of the left ventricle not otherwise explained may be regarded as an almost sure sign of chronic nephritis, represented in fully two thirds of all cases by the contracted kidney in one of its varieties. I will not attempt to explain the absence of hypertrophy in the remaining cases, except to say that it may be due to feeble nutrition in a few. A heart that is too weak or too feebly nourished to take on hypertrophy cannot do so. Some inexplicable supplemental excretion compensating that of the damaged kidney may account for others. I take no note of cases in which valvular disease is associated with the hypertrophy, even though Bright's disease may be present. For in such the valvular disease is competent to produce hypertrophy, and under these circumstances it cannot be regarded as the result of Bright's disease.

The vascular changes associated with Bright's disease are not confined to the kidney, but extend also to the system at large. They are met chiefly in the arteries of middle and small size but may be found in slight degree also in the veins. These changes, grossly speaking, consist in a thickening of the walls of the blood-vessels referred to. Histologically three views have at different times been promulgated. The first of these was by the late Sir George Johnson in 1867, who described the changes as consisting in a true hypertrophy of the *muscular* coat of the arteries.

In 1872, Gull and Sutton,[1] referring to the same changes originally described by Johnson, held that they were in the internal and external coats, and asserted that the muscular coat so far from being hypertrophied was really atrophied. The change in the outer and inner coats they ascribed to the formation of a hyaline fibroid substance, sometimes homogeneous, sometimes fibroid. This, according to them, also extends into the capillaries as a "hyaline granular change." In Germany, Ewald[2] reaffirmed Johnson's views in 1877, ten years after their original promulgation. The third view assigns an important part

[1] Gull and Sutton, "On the Pathology of the Morbid State commonly Called Chronic Bright's Disease with Contracted Kidney," *Med.-Chir. Transactions*, vol. lv., 1872.

[2] *Virchow's Archiv*, lxxi., 1877.

—the most important—to changes in the intima, to which they may be confined, though they may also involve the muscular coat much less frequently; most rarely the external coat. Changes in the intima of an inflammatory character have long been recognized, for example in the early papers of Lobstein,[1] who originated the term *arterio-sclerosis*. Rokitansky[2] and Virchow[3] also recognized them and Virchow first named the process *endarteritis deformans sive nodosum*. According to this third view the vascular changes in Bright's disease are primarily inflammatory in the intima, secondarily degenerate, while the changes in the muscularis are conceded to be degenerate at the outset even when associated with thickening. This third and really oldest view is probably more nearly correct. The hypertrophic thickening of the muscular coat as interpreted by Johnson was a misinterpretation, the hyaline fibroid of Gull and Sutton a result of their technique which included maceration in glycerine. The last three observers did not have the benefit of the most modern technique, which is much improved over that of their day. Among modern observers to whom we are indebted for our more precise knowledge are our countrymen W. T. Councilman[4] and Arthur V. Meigs,[5] whose studies support the later views as to the inflammatory and degenerative nature of the changes in the intima and muscularis.

I should add, however, that there remain those who believe that an actual hypertrophy of the muscular coat does take place, notably, Ewald,[6] Saundby,[7] Haliburton,[8] and Nestor Tirard,[9] the last two being pupils of Johnson; while the appearances described by Gull and Sutton are claimed for arteries not treated with glycerine by William M. Ord[10] and Tirard.[11] The latter says in his recent book on *Albuminuria and Bright's Disease*: "The conditions described by Gull and Sutton

[1] Quoted by Virchow in 1856 in *Gesammt. Abhandlungen*, 1856, p. 498.

[2] Rokitansky, "Ueber einige der wichtigsten Krankheiten der Arterien," quoted in *Schmidt's Jahresb.*, 1854, vol. lxxxi., p. 306.

[3] "Die Lehre von der chronischen Endarteritis," *Virchow's Archiv*, vol. lxxvii., p. 380, 1879, in which he refers to his previous papers on the subject in *Gesammt. Abhandlungen* and elsewhere.

[4] Councilman, "The Relations between Arterial Disease and Tissue Changes," *Transactions of the Association of American Physicians*, vol. vi., 1891.

[5] Meigs, *The Origin of Disease*, Philadelphia, 1897.

[6] Ewald, *loc. citat.*

[7] Saundby, *Lectures on Renal and Urinary Diseases*, 2d ed., Bristol, England, 1896, p. 48.

[8] Haliburton.

[9] Tirard, *Albuminuria and Bright's Disease*, London, 1899.

[10] Ord, in discussion, on "Arterial Disease," in *Transactions of the Association of American Physicians*, vol. vi., 1891.

[11] Tirard, *op. citat.*, p. 189.

frequently co-exist with the true hypertrophy of muscular tissue described by Sir George Johnson."

Next, as to the constancy of arterial changes in kidney disease, precise figures are also wanting. The difficulty is increased by the fact that in the majority of cases no clinical distinction is possible between the arterio-sclerotic form of contracted kidney and the genuine (primary) contracted kidney, which in turn causes arterial sclerosis. In the arterio-sclerotic form the arterial change is always present, being the primary condition, causing both the cardiac hypertrophy and the renal sclerosis. In fact Gull and Sutton declare that all cases of contracted kidney are of this form, as does also Arthur V. Meigs, though maintaining different views as to the essential nature of the arterial changes. I confess, as my experience grows, I find it more and more difficult to decide whether the arterial or the renal disease is primary. And when it is remembered that the same cause may be operating at the same time on the systemic blood-vessels and on the kidney, the difficulties are in no way diminished. Still, cases of contracted kidney are sometimes met where there is no arterial change, others where there is no cardiac hypertrophy, while cardiac hypertrophy is found in cases without general arterial change. Finally, observations are occasionally invalidated by a confusion of simple high tension of the radial with sclerosis of this vessel. Only when a case of chronic interstitial nephritis comes under observation before arterial changes are present, and is watched until the latter make their appearance, can we be certain that it is primary, and while such cases undoubtedly occur, I am not aware of their having been collected.

As to the causes of these cardio-vascular changes,—first, the cardiac hypertrophy, how is it brought about? On one point all are agreed. It can only be the result of extra work, and extra work implies the same conditions as muscular hypertrophy elsewhere, viz., a stimulus to its performance. It is plain that such stimulus may act on the heart muscle directly, or indirectly through increased resistance to the movement of the blood in vessels. The result in either event is increased vascular tension, as shown to the touch by the hard pulse in the radial artery not yet sclerosed, and still more by the sphygmogram of the same pulse. We are supposing the absence of valvular disease. An abnormally stimulating blood, a blood charged with impurities, may act in one or both ways suggested. These were the alternatives suggested to Bright himself. Yet the absolute proof that either is present is wanting. When Johnson's studies seemed to have shown a true hypertrophy of the muscular coat, the explanation was close at hand, a tonic spasm of this coat furnishing the distal

resistance, while the two conditions of cardiac action and arterial spasm reacting against each other would mutually increase the cardiac hypertrophy and the arterial muscular overgrowth. But with the demonstration that the vascular change was a degeneration and not a hypertrophy, this beautiful theory of arterial spasm lost its foundation. We are supposing, too, that as yet no thickened intima is opposing. If we could but find peripheral resistance of another kind, we would still have the required stimulus to cardiac over-action. Accordingly, Traube suggested that it lies in the extensive destruction of the arterio-capillary system in the contracted kidney. This theory was long ago disposed of, when it was learned that ligation of both renal arteries does not raise the blood pressure in the aorta.[1] It was suggested, too, by Ewald, that the required resistance might be found in the capillary area at large—a resistance caused by its unwillingness to admit the toxic blood. I am not aware that such an active power exists. We might escape our dilemma by asserting that cardiac hypertrophy does not take place until an obliterating endarteritis is excited by the impure blood circulating through the vessels. But here we are met with the fact that cardiac hypertrophy and increased vascular tension are at times demonstrable before any disease of the arterial walls is present.

One explanation of cardiac hypertrophy remains which not only fits all cases where there is no demonstrable vascular obstructive lesion, but may also act conjointly with the latter when present, viz., *compensatory cardiac action.* The damaged kidneys being incompetent to relieve the blood of accumulated impurities while the heart is pumping with its usual force, the latter is stimulated by the nervous system to a more forcible contraction, with the result of forcing more blood through the kidney and thus increasing at least the filtration part of its function. This explanation was suggested by Oscar Israel in 1881,[2] and, it appears to me, has not attracted enough attention. Israel based it upon experiment, and in his reasoning therefrom there seems to be no fallacy. These experiments, made in association with Grawitz, consisted in the prolonged feeding of rabbits with increasing quantities of urea. On killing these animals he found not only the kidneys in a state of hypertrophy but the heart also, whence he concluded that the presence of retrograde tissue-products in the blood stimulated the heart to increased activity resulting in overgrowth. Senator,[3] who

[1] It was also objected to Traube's theory that cardiac hypertrophy exists in cases of chronic parenchymatous nephritis where there is no destruction of capillaries, but in this condition also the capillaries are practically destroyed by the forcible compression exerted on them by the exudate in and between the tubules.

[2] *Virchow's Archiv*, lxxxvi., 1881, p. 299. See also Israel, *ibid.*, lxxvii, p. 315, 1879.

[3] *Die Erkrankungen der Nieren*, Wien, 1896, p. 96.

quotes these experiments and conclusions, says such accumulation can explain the hypertrophy of acute and parenchymatous nephritis and the secondary contracted kidney resulting from the latter, but not that of genuine contracted kidney. He does not say why, but presumably it is because he believes, as he states unmistakably elsewhere,[1] that in true contracted kidney no such accumulation of effete products takes place—an unexplainable error. For it appears to me scarcely possible for any one of experience to have failed to realize that such accumulation does take place in contracted kidney, and often leads to the gravest symptoms of uræmia, which appear only when the conservative hypertrophy becomes substituted by the degenerative dilatation.

A very interesting question relates to the hypertrophy of the right ventricle in association with that of the left, which it will be remembered occurs in nearly half of the cases, the remainder being hypertrophy of the left ventricle alone, while hypertrophy of the right ventricle alone never occurs. The coincident hypertrophy of the right ventricle may be a direct result of the effect of the hypertrophy of the left on the nutrition of the heart. The overacting left ventricle throws more blood into the right coronary artery as well as the left, and thus the right ventricle is over-nourished and it hypertrophies. That this should occur in only a part of the cases is quite reasonable.

In conclusion it may be said that no one explanation accounts for all cases of cardio-vascular change found associated with Bright's disease. Two divisions may however be easily made. The easiest accounted for are those which may be included under the heading of the arterio-sclerotic variety, in which irritative substances in the blood, whether autogenous or introduced from without (lead and the poison of gout), excite a primary endarteritis in the whole arterial system including the kidney. In this variety the cardiac hypertrophy and renal contraction are secondary.

The second division includes those cases in which the renal disease is in the true sense primary, and arterial changes are absent when the disease first comes under observation, and yet there is still cardiac hypertrophy. These are best explained on the supposition that the cardiac hypertrophy is compensatory, being invited through the influence of the nervous system. In these, too, there may be added arterial change, invited, it may be also, by the later acquired toxic state of the blood due to defective renal function, but in part also to the primary cause which produced the chronic renal disease, be it lead, gout, syphilis, alcohol, and the like. Finally, hypertrophy of the right ventricle is the direct result of hypertrophy of the left, which excites a hypernutrition in the walls of the right ventricle.

[1] Senator, *op. cit.*, p. 95, and p. 97 a. f.

ON ENURESIS AND "IRRITABLE BLADDER" IN CHILDREN.

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THAT these conditions are not, in by far the greater proportion of the cases, pure neuroses, but manifestations of local changes in the urethra or bladder, many clinicians who have investigated the subject will, I believe, admit. I have chosen to class them under the one heading, for I believe enuresis (the "true incontinence" of Guyon), diurna as well as nocturna, — if we except those cases where it is due to central nervous disease, and those due to polyuria, or foreign bodies in the urethra or bladder, — to be only an advanced stage of those conditions which cause the so-called "irritable bladder," or vesical hyperæsthesia (the "false incontinence" of Guyon). By enuresis, or true incontinence, we mean that condition in which the urine is discharged from the bladder without the knowledge or consciousness of the patient; it occurs, in by far the greater proportion of the cases, as nocturnal incontinence. By "irritable bladder"—better, vesical hyperæsthesia — we mean that condition in which there is an abnormally frequent or increased desire to urinate, the desire occurring at times so suddenly, and being so imperative in character, that the urine is discharged often against the will of the patient, or before preparation can be made to carry out the act normally.

Causes. — Owing to the many difficulties which prevent careful and thorough examination, cystoscopic and endoscopic, in the children affected, — frequently the sufferers are of an age to make such an examination impossible, — no exact data have, as yet, been obtained, and whatever observations have been made rest upon hypothesis. As a result, the little sufferers have, in the past, been subjected to almost all the drugs in the pharmacopœia — and many outside of it — with varying success, chief among them being bromides and belladonna. In

addition, however, in many cases the knowledge or patience of the physician gave out before an accurate recognition of the causative factors was reached, and the matter was dismissed as a "bad habit," and the treatment directed toward improving the *morals* of the child. This has varied from smart beatings upon the nates, or the binding of a stiff brush upon the back at night (so that the stiff bristles might prick the tender flesh and keep the child lying upon its side), to the subcutaneous injection near the anus of ergotin (Henoch [1]). That these last-named measures must have a psychical effect, goes without saying; but there can be no excuse for their existence were an accurate diagnosis made. In view of its widespread prevalence and distressing character, this condition, trivial though it may seem, deserves greater study than it has received in the past.

A glance at the following list of causes, culled from the works upon this subject, must convince one how far the uncertainty concerning the causes has gone:

Central nervous system: Hysteria, neurasthenia, epilepsy, tabes, myelitis, cretinism, idiocy, dreams.

Genital tract: Phimosis, redundant prepuce, preputial adhesions, epispadias, hypospadias, urethritis, vulvo-vaginitis, bacteriuria, cystitis, vesical calculus, vesical tumor, tuberculosis vesicæ, renal calculus, pyelitis, nephritis, urethral obstruction, fragment of calculus impacted in the vesical sphincter.

Other causes: Acute febrile diseases, indigestion, constipation, diabetes mellitus and insipidus, spinal caries, rhachitis, onanism, hypertrophied tonsils, eczema, intestinal parasites, etc.

Guyon [2] says that the etiology of the condition is not clear; that sex causes no predisposition; that the influence of heredity is, however, unmistakable. He believes enuresis to be due to atony of the sphincter vesicæ, and advises faradization of the membranous urethra in the male, or of the urethra just before the sphincter in the female. Desault [3] believes excessive irritability of the bladder to be a predisposing moment; and that, in some cases, the sensation which is caused by the contraction of the bladder and the discharge of urine is so slight that the entire act occurs uninfluenced by the will, and without disturbing the sleep of the child; and that the factors which cause incontinence begin chiefly during childhood, and that, in the course of time, the irritability of the bladder diminishes, while the individual pays more attention to the desire to urinate, and that thus cure usually results. Trousseau [4] believes it to be a neurosis, characterized by the excessive irritability and the abnormally great tone of the bladder-muscle, and, with this idea in view, used antispasmodics (chiefly

belladonna) and hydrotherapy. Baginsky [5] believes it to be a neurosis dependent upon a lesser degree of innervation of the sphincter vesicæ when compared with the detrusor (Ultzmann's view); that in some cases of enuresis diurna, however, it is dependent upon insufficient development of the sphincter; and that, in the most rare instances only, is the cause a hyperæsthesia of the bladder. Civiale [6] believes it to be a sort of overflowing of a full bladder, as a result of the relaxation of the voluntary contraction of the vesical sphincter, and that the desire to urinate is only slightly, if at all, felt during sleep. He advised local treatment, viz., catheterization, the catheter à demeure, and irritating or stimulating injections. Nicolaysen [7] was able, in eight cases of diurnal enuresis, to demonstrate bacteriuria. Imerwol [8] states that a frequent symptom, in boys suffering from gonorrhœal urethritis, is nocturnal enuresis. Personally, I believe the condition, with the exceptions stated above, to be the result of a combination of conditions, the exciting cause being an abnormal increased reflex irritability of the mucous membrane, most marked at the sphincter, and at times, in addition, of the trigonum vesicæ, or sometimes, in the male, of the prostatic urethra, and that the cause of the heightened reflex irritability is to be sought either in a hyperæmia or an inflammation of the vesical neck, the sphincter, or deep urethra, existing either singly or in combination. Unfortunately I have not been able to prove my belief by direct examination of the affected parts in children yet; but the careful endoscopic and cystoscopic, as well as urinalytic, examination of considerably more than one hundred cases of this condition in the adult male and female, as well as the gratifying results obtained in adults and children from treatment based upon this view, have led me to believe that the exciting cause in children is the same as that in the adult, even though the accessory causes may differ somewhat.

Let us digress for a moment, and consider the mechanism of urination. Of the theories advanced to explain the act, that given by von Frankl-Hochwardt and Zuckerkandl [9] seems to be the most logical:

"At first gradual filling of the bladder with urine, then the desire to urinate, which increases in intensity; as a result of this, at first an increase in the already-present tonic contraction of the sphincter, which then relaxes in children and those affected with diseases of the nervous system, and results in expulsion of the urine, while older individuals resist this still further, probably by voluntarily increasing the tonic sphincter-contraction, but at all events by calling the voluntary, auxiliary muscle-fibres into play. When the opportunity to urinate presents, the closure is relaxed, and the detrusor comes into play as an expulsive power."

In the infant, then, up to the time when intelligence and the will come into play, as well as in those individuals whose intelligence remains undeveloped, or is impaired, the emptying of the bladder is a purely mechanical function, dependent entirely upon the inhibition

of sphincter-contraction, through impulses generated in the normal bladder-wall as the result of the distension of the viscus. Similarly, impulses generated in older children, during deep sleep, by over-distension of the bladder, as a result of polyuria, may lead to unconscious emptying of the bladder. But in the normally intelligent child, after the first year or two, where the sphincter is not paralyzed the case is different. In these, the influence of the will comes into play, and the emptying of the bladder occurs normally five or six times during the day, and not at all during the night, precisely as in the adult. Where it occurs much oftener during the day, or at all during the night, where there is an inability to retain the urine, or where the desire to urinate is painful and imperative, we must conclude that the condition is abnormal.

In the normal bladder, infant or adult, the mucous membrane possesses a low degree of sensibility, and the tonic contraction of the sphincter is inhibited voluntarily, or the distension of the viscus and the consequent stimulus to its sensory nerves cause, through reflex action, the inhibition of sphincteral contraction. In the abnormal, hyperæmic, or inflamed bladder, the increased irritability of the diseased mucous membrane and its contained nerve-endings renders them responsive to a far weaker stimulus — a far smaller volume of contents than the normal, or decomposed, or abnormal urine,— and causes the desire to urinate, even before normal distension occurs. Add to this an increased reflex nervous irritability, either as the result of an impairment and depreciation of the general nervous tone, or of hyperæmias or inflammations in the neighborhood, as in the rectum, urethra, kidney, or other pelvic organs (for any condition which causes pelvic congestion is capable of producing vesical hyperæmia), and we have factors abundantly able to account for the frequency and character of the urination. Thus I am inclined to consider most of the long list of causes previously quoted as remote causes only, and to believe that they act only in conjunction with, or as causes of, a hyperæmic or inflamed condition of the deep urethra, or the sphincter, or both. Phimosis, redundant prepuce, preputial adhesions, urethritis, vulvovaginitis, and onanism I count among those factors which cause congestion of the deep urethra; epispadias and hypospadias I consider to be mere chance occurrences in this condition and devoid of any influence in causing it, unless they offer a mechanical obstruction to the flow of urine; bacteriuria, vesical calculus, tumor, or tuberculosis, and pyelitis to be such as cause inflammatory changes at or near the sphincter; acute febrile diseases, menstruation, indigestion, constipation, and intestinal parasites to be causes of vesical hyperæmia;

hypertrophied tonsils, adenoids, eczema, urticaria, and similar conditions to be causes only in that they disturb the sleep and tend to keep the child in a semi-conscious condition. As Trousseau [10] says: "The desire to urinate is, in these cases, felt only slightly, if at all, during sleep,"—that is, the children are only partly conscious of it, and they respond mechanically to the desire to urinate, which desire is called forth by small quantities of urine acting upon abnormal mucous membrane. It is thus that the wetting occurs repeatedly, even though the child be awakened to urinate several times during the night. Similarly the absorption of study, or of play, during the day, may prevent full consciousness of the desire, and the sphincter relaxing, the hyperæsthetic bladder discharges its contents before the little unfortunate has time to heed its call; similarly, fright, shock, laughter, or crying may cause unconscious relaxation of the sphincter. And finally, where he or she *does* become conscious of it, and is forced to respond at frequent intervals, we have the clinical picture of the "irritable bladder."

Diagnosis.—In those children ill with diseases of the central nervous system, marked by paralysis, the incontinence is continuous, occurring day and night, and is marked by no fluctuations. Where the sphincter is *paralyzed*, the bladder retains *no fluid*, this flowing off aside of the catheter. Where the sphincter, or this and the trigonum vesicæ are inflamed, abnormally small quantities of fluid call forth the urgent desire to urinate; this is not the case where they are normal. Aside from this, although touching the membrane of the *body* of the incontinent bladder causes no desire to urinate, slight pressure at the *sphincter* at once calls it forth. Where incontinence occurs during well-marked epileptic attacks, the diagnosis is so clear that it needs no word from me. Where the attacks are less marked, or where they occur during the night, one may at times be unable to get any further history than that of attacks of vertigo, a bitten tongue, short lapses of consciousness during the day, or of twitching, groaning, or grinding of the teeth during sleep, or of heaviness and listlessness on awakening, as signs of the nocturnal epileptic seizures. Where enuresis occurs in the course of neurasthenia, or during dreams, I believe it to be secondary to some local vesical or urethral change.

Phimosis, redundant prepuce, preputial adhesions, epispadias, and hypospadias, I believe, as stated above, to be factors in the condition only in so far as they may cause urethral congestion, or inflammation, and through making infection of the urethra or bladder more easily possible. In urethritis or vulvo-vaginitis, the discharge must, in every case, be microscopically examined, to determine the gonorrhœal or non-gonorrhœal character.

In every case the urine must be examined for albumen and sugar. If turbid, the cause of the turbidity must be determined by chemical and also by microscopical examination. In this manner nephritis, diabetes, phosphaturia, bacteriuria, cystitis, tuberculosis vesicæ, etc., may be determined. Should the urine contain blood, examination must be made to determine the source, whether vesical or renal, and also the cause. Where the age of the patient will permit, or where the condition makes an exact examination and diagnosis imperative, as in cases where the urine is purulent or hemorrhagic in character, or where there is a suspicion of calculus, I consider cystoscopic examination, under the influence of general anæsthesia if necessary, to be indicated and justified, especially since modern skill has given us instruments fitted for use in the bladder of the child, which enable us to make an *accurate* diagnosis of the condition, with the minimum of risk to the patient.

Where onanism is suspected, the child must be most carefully watched. Especially is this the case with little girls, who frequently bring about the desired result through tightly crossing the legs, rocking on the edge of a chair, or even, as in one of the cases which came under my observation, by leaning against the leg of a table.

Phosphaturia, oxaluria, hyperacidity, etc., I believe to come into play as causes of enuresis or vesical hyperæsthesia, only after they have produced hyperæmia or inflammation at or near the sphincter vesicæ —in other words, only after they have destroyed the superficial epithelial layers of the vesical mucosa. In this connection I would refer to the fact that irritating substances (even strong solutions of nitrate of silver) do not, when injected into the normal bladder, produce any sensation at all until the superficial epithelium has been destroyed.

Treatment. — As the diagnosis of the condition resolves itself into a recognition of the local vesical or urethral changes, as well as of the causes underlying and preceding them, so does the treatment resolve itself into that of the local vesical or urethral condition, as well as of the remoter factors.

In considering the treatment, I cannot do better than to refer to the admirable and comprehensive article of our own master, Jacobi,[11] with whose words upon this subject I agree completely. The general health of the child must be looked after and treated, if depreciated, not only by dietary, but also by hygienic and hydrotherapeutic measures. Above all, I would warn against corporal punishment, or anything which savors of brutality, for these can only serve to increase the general nervous depreciation. I cannot believe the condition to be due to innate depravity or slothfulness, as some authors have intimated.

Furthermore, all severe mental strain should be guarded against. The digestive tract must be looked after, and indigestion, constipation, diarrhœa, or intestinal parasites removed by appropriate treatment. The evening meal especially should be light and easily digestible. The genital tract, too, must receive appropriate attention; phimosis must be reduced, adhesions ruptured. Above all, the glans penis and preputial sac must be frequently cleansed, as from this point, in many cases, infections of urethra or bladder have their start. Vulvitis or vaginitis, as well as urethritis, must be treated with reference to their gonorrhœal or non-gonorrhœal character. In the former, I should strongly advise the use of protargol, in ½% solution. If due to other bacteria, then the solution of bichloride of mercury, 1:15,000, will give most gratifying results, the discharge usually fully disappearing in very few days. If bacteriuria be present, the internal use of urotropin, in appropriate doses, combined with irrigations of the bladder, by means of the sterilized catheter, with solution of nitrate of silver, 1:5000, or stronger if the patient tolerates it, will give excellent results. The treatment of cystitis depends, to a great extent, upon the cause; those cases which depend upon the presence of a vesical calculus, or of a foreign body, or upon pyelitis, cannot be cured until the causative factor has been removed. Consequently this must first be sought for and appropriately treated; afterward vesical irrigations with 1% boric acid solution, followed by the instillation of ¼ to 1% solution of nitrate of silver, the latter being, after three to four minutes, allowed to flow off and the bladder subsequently irrigated with boric acid solution, will give excellent results. The polyuria of diabetes or of nephritis must be appropriately treated. That due to the imbibition of large quantities of fluid may be influenced by the restriction of the quantity ingested. I hold it to be a wise precaution to restrict the quantity of fluids taken during several hours before retiring. Onanism must be guarded against by watching the children and by examination for and treatment of the underlying cause; this may frequently be found by watching the playmates. The enuresis which at times accompanies the pelvic congestion of menstruation, preceding or accompanying the flow, is best treated by hot sitz-baths.

Diseases of the respiratory organs, which interfere with proper breathing, such as nasal obstructions, adenoids, or hypertrophied tonsils, must receive appropriate treatment. Similarly also, disturbing, itching skin-eruptions, which act, like those of the respiratory tract, as causes of enuresis, by disturbing sleep.

Concerning the results of faradization of the urethra I cannot speak, as I have not employed it; however, as the urethra which is sufficiently

large to permit of the insertion of an electrode is also large enough to admit the small "children's cystoscope," I should personally prefer to use the latter and obtain some certainty regarding the real cause of the enuresis. Where the size of the urethra will permit, the use of small sounds or of the small-sized psychrophore ought to prove of value, where the condition is due to congestion in the membranous or prostatic urethra. In the female, pencilling of the entire urethral mucous membrane, from and including the sphincter, to the meatus, with $\frac{1}{4}$ to $\frac{1}{2}$ per cent. solution of nitrate of silver, by means of the endoscopic tube, every second day, gives excellent results.

Of drugs, bromides, chloral hydrate, rhus aromatica, belladonna and atropine, ergot, nux vomica, and strychnine have been recommended, belladonna and atropine especially being highly spoken of.

The treatment which has given me the greatest satisfaction, both in adults and children, has been the following: Where the patients were too small to admit of direct local treatment, hot sitz-baths, once or twice daily, the appropriate treatment of accessory causes, the restriction of fluids in the evening, combined with a light evening meal; at night, where possible, the child was laid so that the head was lower than usual, either by laying a pillow under the hips, or, better still, by removing the pillow from under the head, and raising the foot of the bed. Where local treatment was possible, applications in the endoscopic tube, especially directly to the sphincter itself and the mucous membrane of the trigonum vesicæ, or by vesical irrigations and instillations. In this way, proper diagnosis and properly applied treatment will usually effect a cure in from a few days to a few weeks.

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AN EXPERIMENTAL CONTRIBUTION TO THE KNOWLEDGE OF THE TOXICOLOGY OF POTASSIUM CHLORATE.

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Introductory. Our knowledge of the poisonous effect of potassium chlorate is intimately connected with the name of Dr. A. Jacobi. It was greatly due to his writings that the medical profession was awakened to the dangers of this extensively employed drug. Desiring to contribute to the volume to be published in honor of this veteran of medical science, I have selected the study of the toxicology of potassium chlorate as an eminently fit subject for the occasion.

My studies were confined to the experimental side of our subject, and my paper will present briefly the results in two parts: an analysis of the existing experimental data and some of their shortcomings, and the report of some new experimental facts.

I. The nature of the poisonous effect of potassium chlorate was, for a period of about 15 years, the subject of many experimental investigations. Isambert[1] observed in 1875 that, by the addition of potassium chlorate, blood is turned into a dark brown mass; and Jaederholm[2] discovered, one year later, that the chocolate color was due to the formation of methæmoglobin. The general interest in the toxicology of this salt dates back to 1879, when Marchand[3] published an extensive study on the subject. By a series of experiments on dogs and by a study of the post-mortem changes in human poisoning cases, he arrived at the conclusion that potassium chlorate is a blood poison. In the first place, it changes oxyhæmoglobin into methæmoglobin, a product incapable of taking up oxygen. The animal might die in this stage of the poisoning through insufficient oxidation of its vital tissues. In a later stage, also, morphological changes of the red cells take place. The hæmoglobin leaves the cells, and the stromata as well as some normal

red corpuscles break down into fragments. In the further development of the process, these granula accumulate in the kidney and clog the urinary tubes, which leads to methæmoglobinuria on the one hand, and to uræmia with its consequences on the other hand. This view of Marchand found general recognition, and was soon supported by many pathological reports as well as by experimental investigations, among which the excellent monograph of v. Mering[4] is the most noteworthy. This theory, however, was assailed in 1885 by Stokvis,[5] who made an extensive series of experiments on rabbits. Although these animals succumbed to the poisonous effects of the salt, their blood never contained methæmoglobin, neither during life nor soon after death. Stokvis maintained that the appearance of methæmoglobin in the blood is only a post-mortem phenomenon, and that the poisonous effect of potassium chlorate is, in the main, simply the same as that of all other salts — it irritates the kidney and the gastro-intestinal canal. These statements of Stokvis caused quite a lively experimental activity; they were reinvestigated by Marchand,[6] Cahn,[7] v. Limbeck,[8] Riess,[9] Lenhartz,[10] Falk,[11] and others. These investigations have definitely settled that both claims were correct: the blood of dogs poisoned by potassium chlorate contains methæmoglobin even during life, while the blood of rabbits poisoned by the same kind of salt does not show any recognizable chemical or morphological changes.

Since this period of prolific activity, now quite a decade ago, the toxicology of potassium chlorate was not again a subject of experimental investigation. Is it because the problem is solved? Rabbits die promptly from the effects of potassium chlorate. It is definitely established that the blood shows no appreciable changes. Of what do these animals die? Of what do they die when the salt solution is introduced intravenously, or into the peritoneal cavity, when therefore the irritation of the gastro-intestinal canal cannot be the cause of death? The death cannot be due to the depressing effect on the heart by the potassium part of the salt, since sodium chlorate is just as poisonous to animals as the chlorate of potassium. And it cannot be due to an irritating effect of the salt upon the kidney, since there is rarely a nephritis present, and since a nephritis would not kill the animal in so short a time—that is, in less than an hour, as is mostly the case when the potassium chlorate is introduced into the peritoneal cavity. Then we have to admit that the cause of death of rabbits by potassium chlorate is still unknown. But even in dogs is it really an established fact that in all cases death is due to the chemical and morphological changes of the blood? There are experiments on record in which the poisoned dogs died before any changes in the blood took place. Of

what effects did these animals die? On the other hand, we know now, by thousands of instances, that the temporary formation of methæmoglobin within the blood can and usually does pass off without serious consequences. The administration of acetanilid or phenacetine offers such instances. The deep cyanosis often following the administration of these drugs is due to the formation of methæmoglobin within the blood. It is nevertheless a daily occurrence that this cyanosis passes away without any serious after-effects. We also know from numerous instances that the blood manages to get rid of large masses of débris from red blood corpuscles broken down by a variety of causes. Then why should the presence of methæmoglobin or broken down red cells in the blood of dogs after administration of potassium chlorate be absolutely fatal? It seems to me that, in spite of the presence of the chemical and morphological changes in the blood, we are justified in searching for still another factor which perhaps could be considered as the uniform cause of death from potassium chlorate in all animals.

II. In the above described experiments the poisonous salts were administered to the animals either *per os* or by means of subcutaneous, intraperitoneal, or intravenous injections—that is, by all the methods commonly employed in animal experimentation. Some 18 months ago, however, Roux and Borrell [12] introduced a new method of bringing poison into the animal body: it is by intracerebral injections. According to these investigators, the toxins of tetanus and diphtheria when injected into the brain are effective with but about $\frac{1}{20}$ of the dose which is required for effect in subcutaneous injections. Subcutaneous immunization by antitoxin does not protect against intracerebral injections of toxin; intracerebral injection of antitoxin can still save life where the subcutaneous injection fails. They have also found that one mgr. of morphine is fatal when injected into the brain of rabbits, while these animals withstand large doses when introduced otherwise. Injections of heated serum toxin, water, or normal salt solution into the brain have no effect upon the animals. These statements were confirmed by Behring [13] and many other investigators, and this method of injection was extended to the study of the effects of many other substances. Biedel and Kraus [14] have observed a series of typical convulsions immediately after subdural or intracerebral injections of bile and its salts, by which they undertook to explain the convulsions, etc., occurring in *icterus gravis* and cholæmia. Similar convulsions, however, were observed by Bruno [15] after intracerebral injections of morphine, sodium ferrocyanide, and methylene blue; while sodium chloride 4%, sugar, sodium sulphate, and urea 10% had no effect.

In experiments with the introduction of potassium chlorate into the

peritoneal cavity of rabbits, in which the animals died thirty to eighty minutes after the injection, I have observed that death occurred in convulsions, especially those of the expiratory muscles, while ten to fifteen minutes before death the inspirations became weak and shallow. This observation, in conjunction with the fact that the occurrence of convulsions has been recorded in some of the clinical histories [16] and the protocols of experimental poisoning with potassium and sodium chlorate,[17] led me to an experimental study of the effects of potassium chlorate when injected into the brain of rabbits. I made a large number of such experiments and obtained uniform typical results which I am now going to report briefly.

When 3 to 4 minims of a 5 % solution of potassium chlorate are injected into the brain of a rabbit, the animal responds almost instantaneously with violent reactions. In some cases the first few minutes are filled out with all the varieties of forced movement: constant rolling on its long axis, circus movements, etc. Sometimes the scene is initiated by a spasm of the respiratory and laryngeal muscles, interrupted occasionally by shrieks, apparently of a reflex nature. In most cases, however, the first symptom appearing after injection is an opisthotonus, restricted mainly to the upper part of the body and the cervical muscles; at the same time bending the head slightly to the side in which the injection was made. These tonic contractions soon give way to clonic convulsions of the extremities. While the animal is lying on the side opposite to the hemisphere in which the injection was made, all the extremities are making rapid motions, as if the animal were engaged in a rapid run. These motions are soon accompanied by flexions of the head, grinding of the teeth, clonic contractions of the facial muscles, the lids, nystagmus, etc. The corneal reflex is not abolished. These coördinate movements give way later to incoördinate, alternating clonic contractions of nearly all the muscles of the body. The latter convulsions are less intense, and are often interrupted by shorter or longer periods of perfect relaxation of the whole body. In such a period of rest the rapid and shallow breathing is the only indication of life in the stretched-out, perfectly relaxed body. Such periods are often suddenly interrupted by a regular, violent tetanus, which is sometimes the terminal event. This may happen as soon as ten to fifteen minutes after the injection. In this case the tetanus often passes over directly into *rigor mortis*. In other cases the intermissions of rest become longer and longer until all the convulsions disappear, and the animal lies for hours on the same side and on the same spot, with hardly any reflexes. Sometimes death follows in this stage by a gradual cessation of the respiration, but oftener the animal recovers gradually; the paresis of all the muscles

and the stupor of the animal remain noticeable, however, for at least thirty-six hours.

When stronger solutions are employed, the animal succumbs to the attack in a short time, the very violent convulsions are of a tetanic character and of short duration, and terminate either directly in death or in a state of coma from which the animal rarely recovers.

When a few drops of only a 1 % solution are injected, the animal runs about incessantly for perhaps an hour or more, then sinks exhausted into a paretic state which lasts for a few hours; on the next day the animal appears to be perfectly normal again.

I should add that all these effects can also be produced by sodium chlorate; it only requires more concentrated solutions.

These experiments show us that potassium chlorate is a strong poison for the nerve cells, which are first intensely excited, then paralyzed by it. May we not also assume that by introducing this poison into the circulation, a certain amount of it reaches the brain, especially the respiratory centre, in a concentration sufficient to excite and paralyze it? Although we must admit that the solutions injected into the brain contained a greater percentage of the potassium chlorate than the blood of the animal poisoned by this salt ever contains, we must also admit that the circulation reaches the cells of the respiratory centre in a far more intimate way than can be accomplished by our crude methods of simply injecting into the brain, where, according to Bruno, the liquid mostly remains on the surfaces of the ventricles.

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ACUTE CATARRHAL LACUNAR AMYGDALITIS IN THE NURSING INFANT.

By HENRY KOPLIK, M.D., New York,

President of the American Pediatric Society.

"To A. Jacobi, whose life has been spent in stimulating the clinical study of throat affections in infancy and childhood in America, this contribution is dedicated in respectful homage by the author."

THE classifications in the various brochures and text-books of the acute affections of the throat, pharynx, and tonsils in infants and young children are very vague. It will be of value, at the outset of this paper, to define exactly what the writer understands by the various terms he may make use of.

By angina catarrhalis we understand a simple inflammation of the structures which make up the fauces. The anterior and posterior pillars, the tonsil included between these pillars, and sometimes the uvula are also affected. In most of the cases of angina the pharynx will be drawn into the clinical manifestations. The word "catarrhal" is so deeply rooted in our literature and nomenclature that it is best at the present time not to introduce a new term, but to make the old term conform to our present pathological understanding.

Under catarrhal we may very fairly understand any inflammation whose etiological factor is not specific. By specific the writer refers to a specific clinical and anatomical reaction or picture. Thus, diphtheria is a specific inflammatory state due to the presence of a micro-organism whose manifestations take on special clinical characteristics. We would also classify syphilitic angina or gonorrhœal angina as specific. Thus catarrhal angina would include the large class of primary inflammations of the fauces which are due to streptococci or staphylococci The angina due to the streptococcus of scarlatinous throats would not come under this heading. It will be at once seen that the above classification has the advantage of simplicity, and can be made to conform to our advances in the knowledge of this subject in the past decade.

It is clinical and not purely bacteriological, as are the classifications of Monti.

Angina is a generic term, and includes under its heading a number of entities. From a pathological and clinical standpoint we cannot speak of inflammation of the tonsils, or follicular tonsillitis, or lacunar tonsillitis, and not understand that the other structures of the fauces are involved, yet Monti, among other writers, tries to make such a classification. It is not clinical, to say the least.

The tonsil being a very important element in the structures making up the fauces, we can readily see how its inflammation at times will take a leading part in the clinical picture of angina. Thus authors have fastened their attention on this organ to the exclusion of other structures of the fauces. Again, if the above is correct, we can have no angina without a tonsillitis slight or marked.

In speaking of the milder forms of angina catarrhalis, Henoch, in the ninth edition of his classical work upon diseases of children, says that, while children above the fourth year of life are more frequently attacked by this affection than adults, the first years of life are less commonly affected than after the fourth year. Osler, in his *Practice of Medicine*, says tonsillitis is "rare in infancy." Casselberry, in the *American Text-Book*, does not speak of the frequency of angina or follicular amygdalitis in infancy. Holt, in his text-book, simply mentions that follicular amygdalitis is rare in infancy. Lewis Smith says that in infancy catarrhal pharyngitis or angina is apt to escape detection, and also that it is said to be less frequent in infancy than in childhood. Monti makes a distinction between simple angina catarrhalis and angina tonsillaris. Speaking of the latter he says that infants are only exceptionally affected, and that it is most common between the third and eighth years of life. Filatow is not clear as to frequency. In one place he says that acute inflammations of the throat are common in childhood, and not recognized below five years because children at such age do not complain of pain in the throat. On the next page, however, he says the simple angina catarrhalis is quite rare — in fact, rarer than the follicular or lacunar forms (Semiotik).

In his lectures on infectious diseases, Filatow emphasizes the necessity of examining the throat in every case of sick infants, inasmuch as sore throats may run their course latent without marked symptoms, a point which will be further illustrated by the writer under description of symptoms.

In the following table I have taken the cases below 12 months of the total number of cases of angina catarrhalis from my clinic books

for six consecutive months, including all cases of lacunar amygdalitis below the age of ten years.

CASES BELOW TWELVE MONTHS OF AGE OF 1284 CONSECUTIVE CASES OF ANGINA CATARRHALIS BELOW THE AGE OF TEN YEARS, INCLUDING CASES OF ACUTE CATARRHAL LACUNAR AMYGDALITIS, TABULATED.

Months.												
1	2	3	4	5	6	7	8	9	10	11	12	
0	3	2	3	8	9	7	2	3	3	8	5	January.
0	0	3	1	4	4	3	3	3	5	1	4	February.
1	2	4	0	11	5	7	11	2	4	3	20	March.
0	2	1	1	2	10	3	1	6	5	4	16	April.
1	1	2	3	4	4	2	2	1	4	2	11	May.
3	2	6	2	4	4	1	1	5	1	3	9	June.
5	10	18	10	33	36	23	20	20	22	21	65	

Total 333, 12th month inclusive.
" 0 to 5th month inclusive, 76.

It will be seen that these inflammations of the throat are by no means uncommon, that they form fully 25 per cent. of all the cases of angina below the age of ten years, that from birth to the sixth month these conditions are least common, forming only a little more than one third of the throat inflammations below the age of one year. After the sixth month the frequency of anginas increases, to attain greatest frequency at the twelfth month of life. I do not think that the infrequency of angina shown by the figures between the ages of 0 and 5 months is to be entirely attributed to immunity at that tender age, but rather to the unwillingness of mothers to bring such young sick infants out-of-doors to a clinic. This is especially so of infants below the age of two months. I feel that if opportunities were offered to examine more throats at this age a greater number of cases of angina would be found than appears from a study of this table. I do not claim that the above table of frequency is final. All such statistics must vary with place, locality, time of year, and epidemiological influences, and class of material. It tends to prove uncontrovertibly the frequency in the nursing period of acute primary catarrhal affections of the throat, also the necessity of examining the throats of such patients as a routine procedure.

Once inflamed, the tonsils will present varying pictures according to the severity of the inflammation and the amount and character of the inflammatory products present. The tonsil, being a lymph node of active function as has been shown by Stöhr, is even in conditions of health undergoing interesting changes. These changes consist in a constant throwing off in the depths of the lacunæ of lymphoid cells

which are coming to the surface to pass away in the faucial secretions. If an inflammation is present these processes become abnormally active. We have in severe forms of inflammation of the tonsil a plugging of the lacunæ with bacteria and leucocytes. At the surface these plugs appear as yellow spots or streaks. There is nothing specific in this appearance, it is only a clinical picture. Pathologically speaking, any inflamed tonsil must be inflamed in its lacunæ. The reason all forms of tonsillitis of a simple catarrhal form do not show the lacunæ clinically may depend rather on the nature of the products of inflammation. In one case they are adhesive, yellow, and plugging up the lacunæ; in the other, being more fluid and passing away at the surface of the tonsil, leaving the lacunæ clear and unmarked clinically.

I have gone into the topographical data because it is a common idea among practitioners that a lacunar amygdalitis or tonsillitis has something specific about it. This is scarcely so. The tonsil being made up in folds, or lacunæ, it is evident, if what I have said be true, that most forms of tonsillitis are pathologically and anatomically lacunar if they are not so clinically. Thus, to speak of an angina catarrhalis or angina tonsillaris, or angina lacunaris streptococcica or staphylococcica, does not aid us any in lucidity; it only complicates a very simple clinical entity.

The Normal Structure. Some writers wishing to emphasize the presence of the enlarged pathological tonsil have said that normally there are no tonsils. This is not only incorrect on its face, but it also ignores an organ whose functions must be of very great importance, even though it is small in size normally. If we look into the mouth of a very young infant with normal fauces, we find the structures of a very faint bluish-pink color. There is no suspicion of redness either of the pillars of the fauces or uvula. The tonsils are seen as exceedingly small, pale bluish-pink bodies concealed or included in the anterior and posterior pillars of the fauces. The normal tonsil is never longer though flatter than a split pea. There is no redness anywhere. In older infants from about the seventh to the twelfth month we may see normally on the anterior pillar of the fauces small transparent miliary bodies which look like drops of dew. These bodies are normal to the infant's throat; they are just in front of and above the situation of the tonsil. These are generally two or six in number on either side; they are collections of lymph-adenoid tissue. Their importance will be shown in abnormal states. It is not always easy to see the tonsil in the normal state, unless we make the infant gag a little. At such times the tonsil is seen to be thrown into irregular folds on the surface, with intervening depressions. In these depressions, the

outward evidences of the lacunæ, small curds of milk may frequently be seen. When examining the infant the little patient will gag. At such times the pillars of the fauces, if the tonsil and parts are normal, remain flat, they do not bulge at the situation of the tonsil. This is important, for in the abnormal state they will bulge as the infant gags, thus giving evidence of the enlarged body of the tonsil behind the palate folds. To complete the picture of the normal fauces, the arch in the fauces reveals the posterior pharyngeal wall, which is smooth, whitish-blue or bluish-pink, and glistening in aspect.

Abnormal Clinical Appearances. If there is an inflammation, slight or marked, of the structures of the fauces mentioned above, the first striking symptom is a distinct red discoloration of the anterior pillars of the fauces, the uvula, and posterior pharyngeal wall. As we examine the fauces and make the infant gag, we see that at the situation of the tonsils, though still covered by the anterior pillars of the fauces, there is a distinct bulging the size of a bean or larger. The tonsil is swollen and reveals its presence beneath the pillars of the fauces. A closer examination shows in nurslings of even tender age that the tonsil is enlarged. Even at the surface it is more prominent, the folds on the surface are larger and swollen. In the vast majority of cases a close inspection reveals small, exceedingly fine, yellowish-white points. These points may in some cases look like very fine scratches of a yellow tint. These are the lacunæ filled with the inflammatory product. In older infants, six to twelve months, these may be still more prominent. The yellow or yellowish-green spots are quite large; the tonsil is much greater in size. We may see even evidences of ulceration on the surface. In some infants, ninth to twelfth month, we may have a film of secretion of a yellow or greenish color on the tonsil. The small miliary lymphoid bodies described above on the anterior pillars of the fauces, just in front of the tonsils, are in the inflamed state, large and red and can be very plainly seen to be twice the normal size; sometimes we have them break down into minute ulcerations, herpes-like vesicles preceding the ulceration (herpes of the tonsils, old nomenclature).

I have described very minutely the clinical appearances because they will be overlooked even by a careful examiner if not sought for, as in the vast number of cases the lacunar appearances are small and not so numerous as to number of spots as in the older tonsil. The appearances are so small and sometimes so well hidden behind the pillars of the fauces that unless we study them as the infant gags they will escape notice.

Of the remaining structures we should mention the uvula, which may not only be inflamed but relaxed and hang down abnormally low.

Just above the uvula the soft palate may present a punctate redness. The posterior pharyngeal wall is reddened and the normal collections of lymphoid tissue are enlarged and red, in some cases giving a beaded appearance to the surface of the pharynx.

I have been exact, and those who will study these appearances as I have for years will easily confirm the above. Thus, in nursing infants who have a simple catarrhal inflammation of the throat, it is not difficult to absolutely fix the fact that in all cases the tonsils are inflamed. In the great mass of cases it is easy to discover a process of a pathological nature which is the exact clinical counterpart in miniature of the lacunar amygdalitis in the older infants above the first year of life.

Etiology. If we exclude diphtheria, the infant is not only very susceptible of infections of all kinds, but is constantly exposed to them. Epstein and his pupil, Fischl, have well elaborated this part of our knowledge. We have only to pass in review the newly born infants which we have seen suffering from the various forms of stomatitis to grant that the mucous membrane of the infant's mouth is easily wounded and thence infected. The mother is constantly interfering with the mouth of her infant either by introducing the finger unnecessarily or cleaning it too harshly. The breast or bottle nipples are also harbingers of infection. The mother herself may have an acute or subacute angina. She exposes the infant to infection in many ways. My experience shows that not only does the infant succumb to these infections, but the simple infections which I will describe are the most common to be found in infancy, instead of being, as the majority of authors say, uncommon.

I have examined bacteriologically a number of inflamed throats of infants which show the clinical picture of lacunar amygdalitis. This is not an easy task. To get the secretion from the tonsil in some cases requires a very steady hand and good eye, in the cases in which the lacunæ are small. In some cases a sterilized spud was used to obtain the secretion, in others, a platinum loop. Care was taken not to contaminate the culture with saliva. In many cases, in fact, it is unavoidable not to get some surface secretion of the tonsil on the spud. We did as well, however, as the circumstances of the cases allowed without going so far in our scientific ardor as to shock the parents of the patient.

1. Male, 12 months old, follicular amygdalitis, appearances well marked, 5 days ill.
   Bacteriological examination: streptococci and staphylococci.
2. Male, 7 months old, sick 2 days, follicular amygdalitis marked.
   Bacteriological examination: streptococci.
3. Male, 10 months of age, distinct follicular amygdalitis.
   Bacteriological examination: principally streptococci.

4. Male, 4 weeks old, distinct lacunæ.
   Bacteriological examination : staphylococci.
5. Male, 8½ months of age, distinct lacunar amygdalitis.
   Bacteriological examination : staphylococci.
6. Female, 7 months of age, distinct lacunar amygdalitis.
   Bacteriological examination : staphylococci.
7. Male, 7 months of age, distinct lacunar amygdalitis.
   Bacteriological examination : staphylococcus aureus.
8. Female, 6 months of age, distinct lacunar amygdalitis.
   Bacteriological examination : staphylococcus aureus.
9. Male, 9 months of age, distinct lacunar amygdalitis.
   Bacteriological examination : staphylococcus aureus.
10. Male, 5½ months of age, lacunar amygdalitis.
    Bacteriological examination : staphylococcus aureus.
11. Male, 9 months, lacunar amygdalitis.
    Bacteriological examination : streptococcus.
12. Female, 6 months old, lacunar amygdalitis.
    Bacteriological examination : staphylococcus.
13. Female, 1 year old, lacunar amygdalitis.
    Bacteriological examination : streptococcus.
14. Male, 6 months old, lacunar amygdalitis.
    Bacteriological examination : staphylococcus aureus.
15. Female, 13 months of age, lacunar amygdalitis.
    Bacteriological examination : Roux coccus.
16. Male, 9 months of age, lacunar amygdalitis.
    Bacteriological examination : streptococcus.
17. Male, 1 year old, marked lacunar amygdalitis.
    Bacteriological examination : streptococcus.

The above is only a small fraction of the material which has passed under my personal observation. It is evident that nothing was to be gained by heaping up material. The above will show beyond question that in a large clinical material it is not difficult to find cases of lacunar amygdalitis among the nurslings. Our views, therefore, as to frequency must be considerably modified. It is not by any means a rare affection at the nursing period of life. It is very commonly met with. Either its frequency has hitherto been overlooked or the material coming to the various writers was not sufficiently abundant among the nurslings, or various clinicians have not taken care to look for this affection.

We are struck by the infrequency of diphtheria infection among these nurslings. I have among my notes a record of only one case, an infant 10 months of age, in which I have been able to find undoubted, virulent, Loeffler bacilli. In this infant there were placques of membrane on the tonsils. A. Jacobi has pointed out, many years ago, the infrequency of diphtheritic infection in the new-born and nursling. He records one such case in the new-born. Feer records two infants, 2 to 6 months of age, with diphtheria, and shows by tabulation that of

4240 cases of diphtheria, 112, or 2.5%, were under one year of age. Baginsky shows the frequency of diphtheria up to the 6th month of life, ½ of 1%. Escherich has endeavored to explain the so-called immunity of the new-born and nursling from diphtheritic infection as something inherent in the blood serum of these infants. The simple infection, it will be seen from the above list, is mostly due to streptococcus and staphylococcus aureus.

Symptomatology: If we try to formulate the symptoms of lacunar amygdalitis in these nurslings we find in some cases there is:

Fever. This fever may be quite high, 104° or more. In other cases in which the lacunar amygdalitis was very marked, there was an absence of any febrile reaction. This occurred in a male infant one year of age, in whom the tonsils were the size of a small bean; the lacunæ were plugged with large plugs of yellow-green purulent material on both sides. There was no glandular enlargement and very little swelling at the angle of the jaw. The lacunar plugs, removed and examined, contained streptococci. The first day the mother said the infant would not nurse. Rectal temperature, 99½°. Second day, tonsils not covered by so much greenish deposit but lacunar amygdalitis still very marked. Rectal temperature, 98°. In this case the temperature was subnormal, if anything. This only confirms what is known, and what has been so ably pointed out by R. Fischl, that infants at the breast react very slightly even to serious infections. Infections which in the adult would cause marked reaction, cause little or none in the infant.

Pain in Fauces: Nursing. A mother will frequently come and tell us that the baby does not nurse. This condition is undoubtedly brought about by the pain which the infant experiences in swallowing.

Diarrhœa. The intestinal movements in these cases may be disordered. We might expect this, not only from the presence of the fever, but also aside from fever, by direct infection of the gut by the faucial secretions swallowed by the infant. In other words, the infant infects itself. The movements may be green, of a bad odor, diarrhœal, several being passed during the height of infection, of a watery character.

Glandular Swelling at the angle of the jaw may not be present, or the glands may be swollen to a great size. Thus many cases of adenitis suppurativa at the angle of the jaw, in infancy especially, will be found to have been preceded by an inflammation of the fauces. Nay, more, the investigations of retro-pharyngeal abscess certainly point toward such a possibility (Koplik). Otitis is not an uncommon complication.

Duration. The infant who is a sufferer from acute lacunar amygdalitis will often puzzle the physician. It is difficult to believe an amygdalitis in some cases very slightly marked will cause the infant to be ill, refuse nourishment, and cause restlessness, diarrhœa, and much constitutional disturbance for three or four days, yet such is the undoubted history of many of these cases. The writer has been able to confirm this in cases in private practice where all sources of error had been carefully eliminated. On the other hand, a vast number of the above cases run their whole course in an obscure latent manner.

The necessity of examining, as a routine procedure, the throat of every nursling brought to us is thus emphasized.

NASO-PHARYNGEAL DISEASE IN PEDIATRIC PRACTICE: A CLINICAL STUDY

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NASO-PHARYNGEAL disease in pediatric practice may be viewed from one of two standpoints — the specialist's or the general practitioner's. The former is apt to see the cases late, when serious secondary troubles may have developed; the family physician, on the other hand, is more likely to be consulted at an early date. It is therefore important that the possible evil influences, direct or indirect, exerted by naso-pharyngeal troubles generally, and adenoids in particular, should be kept in mind — otherwise the treatment will be symptomatic and palliative, rather than radical and curative.

The symptoms vary with the individual. In one the brain receives the brunt of the attack, in another the chest, in others circulatory or digestive disturbances are manifested, and so on. In some the relationship is evident; in others a careful study only will clear up the case. Much may be accomplished in the way of prophylaxis by a correct and early diagnosis.

The following, taken from Jacobi's masterly, instructive, and scholarly article, "Some Preventives," is suggestive and will serve as our text: "Nasal catarrh, with its hyperæmia and soreness of the mucous membranes, predisposes and causes chronic hypertrophy, adenoid growths, tumefaction of submental and submaxillary lymph-bodies, invasion of diphtheria and tuberculosis, and occasionally meningitis." (*Philadelphia Med. Journal*, Dec. 10, 17, 24, 1898.)

It is not to be inferred from the above, that adenoids are the result of repeated attacks of nasal catarrh in all cases. In numerous instances, particularly when occurring in families free from syphilis or tuberculosis, the lymphoid hypertrophies must be regarded as the local

manifestation of a constitutional dyscrasia, to which the term lymphatism has been applied.

In quite a number, the trouble is congenital or shows itself within the first few months after birth. As lymphoid hypertrophies in the upper and middle pharynx are frequent in children, it seems but logical to conclude that the enlargement of the pharyngeal tonsil in many cases is primary, and the catarrhal condition of the naso-pharynx, particularly when attended by a semi-purulent discharge, secondary — an effect and not the original cause. Our work will be facilitated and the ground cleared for subsequent discussion in detail, if at this point we refer to the functions of the nose and indicate briefly the anatomical relations of the naso-pharynx.

The main functions of the nose are:

(*a*) Respiratory,

(*b*) Olfactory,

(*c*) To give resonance to the voice,

(*d*) And to act as a regulator of the aëration of the middle ear, and, we may add, of the accessory air chambers or sinuses in the frontal, maxillary, ethmoidal, and sphenoidal bones.

Two conditions,—patency of the nose and throat, and a healthy mucous membrane,—are essential to the proper performance of the work. Disease, with a greater or less degree of stenosis, shows its evil effects in many ways, to be discussed later on.

The naso-pharynx serves as a common area of air communication between five openings. The Eustachian tubes, one on either side, posterior to the nasal choanæ, ventilate the middle ear. The acuteness of hearing depends upon the patency of the openings with free nasal respiration. The posterior nares also open into this space. They act as the normal channel for the passage of air through the nares to the lungs. Unobstructed nasal breathing is essential to the proper ventilation of the accessory sinuses of the frontal, superior maxillary, ethmoidal, and sphenoidal bones. Finally, at the lower portion, communication is established with the oro-pharynx.

As a pathological entity encroaching upon or invading this space, we frequently meet with a hypertrophied condition of the lymphoid structures (Waldeyer's Tonsillar Ring). The symptoms are local and general. Some are caused by pressure, others are inflammatory in character, and many are the result of anatomical changes more or less permanent.

A discussion of the topic may appear trite to the specialist—it cannot be told too often to the general practitioner. Specialists, as a rule, do not see the cases early; the general practitioner, on the contrary,

is frequently consulted at a time when a recognition of the trouble enables him to ward off many outward evil effects by proper local treatment, operative or otherwise.

Though naso-pharyngeal troubles are very common, in general practice, unfortunately, they are frequently overlooked, treated lightly, or dismissed with a few general directions. This is a serious error. Parents must not be led to believe that the child will outgrow the disorder, or that the symptoms will disappear about the time of puberty.

Advice of this sort, with neglect of appropriate measures, is certain to be detrimental to the mental and physical welfare of the patient. The popular belief, that operations upon the tonsils, etc., may be followed by defects in speech or imperfect development of the genitals, must be combated. Parents often refuse operative interference, until assured that no evil results will follow in this respect.

The family physician does well to remember that his duties are not confined to the treatment of an individual case or disease. Children under his care ought to be regarded as his wards from a medical standpoint. With a history of recurring attacks of nasal catarrh or mouth breathing, the dangers should be made clear to the parents. Unnecessary delay or procrastination must be avoided.

The attendant should bear in mind that the effects are not altogether local. Disturbances, cerebral and nervous, due to the obstructed blood and lymph circulation at the vault of the pharynx and base of the brain, are frequent. Deformities of the chest, bronchial and pulmonary inflammations, are common, as well as recurring attacks of catarrhal croup.

The general circulation is interfered with, respiration, digestion, etc., disturbed, and dyspnœa may be present. The poorly developed muscles, with lowered vitality in general, lead to chronic invalidism or render such patients an easy prey to acute disease.

In addition, enlarged glands at the angle of the jaw, repeated attacks of nosebleed, acute and chronic bronchitis with emphysema and asthma, broncho-pneumonia, large bronchial and mediastinal glands, are frequently secondary to a morbid state in the naso-pharynx. The paroxysmal nocturnal cough, quite common in children, distressing and alarming in character, disappears when the nose and throat are treated. The special senses, taste and smell, are more or less impaired in older children. The voice is altered and assumes a nasal character. Inability to pronounce the letters *m* and *n* and in some cases stuttering exist.

Diseases of the eye may be reflex or arise from a direct extension of the process in the nose. Most commonly there is direct extension.

Deformities in the nasal passages, acute or chronic catarrh, and adenoids give rise to affections of the lachrymal sac and conjunctiva.

Pupillary changes, photophobia, disturbed accommodation, strabismus, blepharospasm, etc., are at times of reflex origin. A normal mucous membrane is the best safeguard against the onset of a number of infectious micro-organisms. The invasion of diphtheria, tuberculosis, and now and then meningitis, is favored by an abnormal condition of the nasal and pharyngeal mucous membrane. The best preventive, therefore, is to keep the mucous membrane in a healthy state. The eloquent appeals in favor of a routine naso-pharyngeal toilet have aided somewhat in popularizing the method. In the tenement districts, where most necessary, the precautions are imperfectly employed or wholly neglected. In this connection, it may be stated that, when a child with adenoids and associated nasal catarrh contracts diphtheria, an extensive surface is apt to be involved. The type will be severe, the progress correspondingly grave. On the other hand, children who "take cold" easily, who present but few evidences of lymphoid hypertrophy up to this time, often develop decided symptoms of obstructed nasal breathing after an attack of diphtheria, scarlet fever, or measles—at times, in spite of carefully conducted nasal toilet during the course of disease.

Small painless glands at the angle of the jaw, about the size of an almond, are common. Though frequently mistaken for tonsils, this is an error. They are due to infection from the naso-pharynx, and point to the presence of adenoids or a moderate degree of nasal catarrh. If an exacerbation of the latter takes place or an infectious disease is superadded, the glands begin to swell and become more or less painful. Under appropriate treatment with nasal injections and cold applications externally, the process subsides and resolution takes place. In other instances suppuration occurs, either glandular, periglandular, or both. Now and then the capsule becomes thickened and the process remains quiescent; sometimes caseation takes place or calcareous or fibroid degeneration occurs. Other chains may be involved, the process extending downwards to the bronchial glands. The chief danger, however, lies in the tendency to become tubercular. In the latter case, the process may remain local, infect other glands and tissues in the vicinity, or general tuberculosis may result eventually.

Surgeons, recognizing the danger, advocate and practise the removal of enlarged or tubercular cervical glands. Yet adenoids and large tonsils have been allowed to remain, to serve as a nidus for subsequent infection. They, as well as the external glands, ought to receive surgical treatment.

A large proportion of ear troubles, from 60% to 75% according to different authorities, are secondary to diseases of the nose and throat.

Adenoids, in particular, constitute an all-important etiologic factor. In nearly every case ear disease is certain to follow, and no time should be lost in advocating their removal as a prophylactic measure. Clifford Allbutt says the very worst degrees of depressed ear-drums are found in those affected with large growths. Deafness, deaf-mutism, and ear disorders in general are benefited at times by local treatment of the throat. In the course of the exanthemata and other infectious diseases, suppurative otitis with perforation is very apt to develop whenever a prior inflammatory irritation or congestion of the naso-pharynx is present. The danger is increased if the pharyngeal or faucial tonsils are hypertrophied. Otitic troubles arise in several ways.

The Eustachian tube may be occluded with mucus, the pressure of adenoids against the orifice may cause its obstruction and thus interfere with the proper ventilation of the middle ear, or the catarrhal inflammation may extend through the tube and involve the delicate structure of the ear.

Trousseau, years ago, and others since then, have called attention to recurring attacks of erysipelas of the face in chronic aural or nasal catarrh with erosions of the skin. New outbreaks are avoided when, as a prophylactic measure, the primary condition in the ear, nose, or throat is relieved. A few cases of this kind have come under our observation at the Vanderbilt Clinic. The same is true of dermatitis and eczema under analogous conditions.

A word as to general diseases accompanied by local throat or nasal symptoms. In tuberculosis, syphilis, and rheumatism,[1] and in the acute infectious diseases, the general characteristics are such that the nature of the local condition does not remain in doubt for any length of time. Now and then some difficulty may be met with in diagnosis.

Anatomists have clearly demonstrated the direct lymphatic communication between the vessels in the naso-pharyngeal mucous membrane and those at the base of the brain. Bacteriologists have reported the presence of micro-organisms in the nose and throat similar to those found in many cases of meningitis. Clinical observations show that the different varieties of meningitis are most commonly observed between the ages of three and five years, at a time when naso-pharyngeal troubles are very common. The intimate lymphatic connection

[1] Since the above was written, Dr. Packard, in the "Wesley M. Carpenter Lecture," discusses in an able manner "Infection through the Tonsils," especially in connection with acute articular rheumatism.

referred to, and the identity of the micro-organisms in the naso-pharynx and those found in a large number of cases of meningitis, tend to explain the etiology of many heretofore obscure inflammations of the brain and meninges.

A general infection by way of the blood must be distinguished from a local infection arising from some region in the neighborhood of the skull. A frequent mode (besides the one referred to above) is through the Eustachian tube to the middle ear and thence to the cranial cavity. As a result, thrombosis, sinus-pyæmia, inflammation of the meninges and brain, with or without abscess, are not infrequent.

Growth in general is more or less interfered with in many instances. Ewing, in an excellent article directing attention to the work done abroad, presented additional facts showing the diminished power of resistance, with the liability of sudden paralysis of the heart, in many of these patients.

Furthermore, a number of cases of sudden death during anæsthesia for the removal of adenoids have been collected by Hinkel.

Deformities of the thorax, due to adenoids, are met with, though it should not be forgotten that other factors are usually associated. The worst cases occur in rhachitic subjects, particularly when bronchitis and pulmonary inflammation have been of frequent occurrence. The deformities vary in degree from the flat chest of the milder to the "barrel-shaped" and "pigeon breast" of the advanced type.

In seeking an explanation, it may be interesting to refer to the effects of nasal obstruction upon respiration, and to note the difference in the physical character of the air when it reaches the lungs in a normal manner through the nares, or abnormally by the way of the oro-pharynx. Inspired through the nose, the air is warmed, filtered, and moistened; in addition, further modifications occur from an interchange of gases between the blood and the atmospheric air.

When breathing is carried on through the mouth, these changes do not occur, and the air, not being filtered, warmed, or moistened, acts as an irritant. Consequently the delicate structures of the larynx, bronchi, etc. (rendered more susceptible because of the chronic catarrhal inflammation of the nose and throat), readily become inflamed. As the distal portion of the lungs do not fully expand under such conditions, the external atmospheric pressure being greater, the chest wall sinks in and deformities result. The degree varies according to age, the condition of the bony structure of the chest wall, the development of the muscular tissues, the presence of bronchitis, and the amount of existing pulmonary collapse or deficient expansion. We are all perfectly familiar with the difficulty and discomfort experienced in breathing when

afflicted with a cold in the head. The respiration becomes labored, and the lungs expand imperfectly for the time being.

In the case of the infant or child, the condition is more or less permanent, depending upon the degree of stenosis and the presence of acute or chronic catarrh. The breathing is superficial, and the effects are more severe and lasting. Let any one attempt the simple experiment of breathing through the mouth for a short time, he will quickly realize the discomfort and fatigue, the dyspnœa, sense of imperfect expansion, and the feeling of weight upon the chest.

In mild cases, in the young, the lungs, expanding imperfectly, allow the thoracic walls to fall in, causing a shortening of the antero-posterior diameter. The chest becomes thin and flattened, the intercostal spaces are depressed, and the infra- and supra-clavicular regions retracted. The Funnel Breast (Trichter-brust), characterized by a funnel-shaped depression at the lower portion of the sternum, certainly, in some cases, is secondary to the nasal obstruction. It has been my good fortune to see a few in the process of development.

My experience accords with Osler, who says: "During inspiration, the lower sternum was forcibly retracted, so much so that at the height the depression corresponded to a well marked 'Trichter-brust.' While in repose the lower sternal region was distinctly excavated." A similar state of affairs was observed in an infant with a syphilitic affection of the nasal mucous membrane. The deformity disappeared as the nasal symptoms improved under antisyphilitic treatment.

In marked cases associated with rickets, the chicken or pigeon breast is observed. The sternum is prominent, particularly at the junction of the first and second portion, the ribs project anteriorly, while laterally, above the diaphragmatic or rhachitic groove, the chest is depressed, giving a triangular shape to the thorax. In advanced cases, the chest is almost fiddle-shaped. In a well marked instance in a child eighteen months old, presented at one of our classes during the "Practical Course," it was surprising how quickly the deformity was remedied, when the patency of the naso-pharynx was restored. The "Barrel Chest" is not infrequent, and occurs in those who are afflicted with chronic bronchitis, emphysema, and asthma. The neck is short, and round shoulders with or without scoliosis may be present.

In the absence of other causes, Coolidge believes that some of the atypical orthopedic deformities may result from a lowering of the general nervous vitality, frequently seen in patients with adenoids. Bilhaut found voluminous adenoids in many cases of scoliosis, removal of which at an early date brought about cure. Whatever the relation may be, it is important to secure pulmonary expansion in such cases,

as the cure or improvement of the scoliosis is facilitated by furthering the development of the muscles and establishing good nasal respiration.

A practical point in hastening the cure of empyema may be incidentally referred to in this connection.

In a few cases of empyema in mouth-breathers, curetting of the naso-pharynx, by favoring pulmonary expansion through improvement in the breathing, caused the obliteration of a small cavity or sinus, thereby avoiding a secondary operation upon the costal walls. In the same way, the associated lateral curvature rapidly disappeared when nasal respiration was established.

Snuffling in infants with retracted root of nose is of such evident import that even the tyro in medicine gives a correct interpretation. The nasal deformity should not be confounded with a similar state in cretinism and some forms of idiocy. The change in the appearance of the face, due to long-existing mouth-breathing, is characteristic, and admits of a ready explanation. The dropping of the lower jaw, due to a functional loss of tone in the muscles, adds to the length of the face, the latter appearing longer because of the deficient development of the superior maxilla.

The proper ventilation of the accessory sinuses or air chambers is interfered with by the naso-pharyngeal obstruction. As a result, the blood supply is modified, normal growth of the bones does not occur, and expansion is retarded. The anæmia and malnutrition, in consequence of the accompanying digestive and circulatory disturbances, leave their impress upon the face and give the drawn appearance to the eyes and mouth. The facial muscles are poorly developed, and the pinched nose or distended alæ add to the deformity. These changes, taken in connection with the mental state, give rise to the characteristic physiognomy.

A high-arched palate, with narrowing of the transverse measurements of the jaw, presenting a pointed appearance in front, with resulting contraction of the alveolar process, crowding and even rotation of the teeth on their axis, is frequent. The absence of the support of the tongue and increased atmospheric pressure upon the roof of the mouth, in consequence of the buccal breathing, explain the deformity. The gothic-shaped palate in turn crowds the septum, causing a deflection, and thus adding another factor to aggravate the inconvenience of the original trouble.

The teeth show a tendency to early decay, particularly the molars. In some cases, stomatitis and gingivitis occur, persisting until the growths are removed. The breath is more or less offensive, the odor being caused partly by the bad teeth and partly by decomposed

secretions, etc. Imperfect mastication, the rapid bolting of food, and the general anæmia keep up and intensify the dyspeptic symptoms.

In younger children, particularly under a year, after exhausting disease with pronounced muscular weakness and relaxation, there is an additional danger, due to the tendency to falling back of the tongue and possible asphyxiation in consequence—particularly if the patient is allowed to sleep upon the back. Such cases must be carefully watched, and must be kept lying on the side. Strychnia and good diet soon restore the muscular tone.

Older persons frequently complain of shortness of breath. Talking, going up-stairs, or rapid walking produces dyspnœa and palpitation. A careful examination shows that the symptoms are due to the nasal trouble, and not to heart disease.

Naso-pharyngeal obstructions induce abnormal breathing, anæmia, disturbed sleep, and a variety of nervous manifestations. The disposition is altered, the children become fretful or sullen, the memory is defective, and, apart from the impairment of hearing, such patients are inattentive, backward, and dull.[1] In cases in which the growths have existed for a long time, the process may cause anatomical changes in the meninges and brain, with resulting idiocy.

Headaches, often of a low grade, limited to the forehead and temple, may be accounted for by the retention of morbid products and obstructed circulation. Attacks of night terrors, walking in sleep, morbid dreams, melancholia, and other evidences of disturbed cerebral functions may occur.

The mental and nervous phenomena are of extreme interest and importance. The question has been studied by Wells in an able and exhaustive article (*Amer. Jour. Med. Science*, Dec., 1898), from which the following is quoted:

"Since we are dealing especially with psychopathic phenomena, how, we may inquire, can an obstructive lesion of the nose interfere with the cerebral functions? Briefly, by (*a*) alteration and impoverishment of the general, and secondarily of the cerebral, circulation, from the overcharging of the blood with CO_2 and the diminished supply of O, which are the necessary results of deficient aëration; (*b*) interference of the blood-supply of the brain by the lesion in the nose; (*c*) hindrance to the outflow of lymph from the brain. It has been shown that the subdural and subarachnoid lymph spaces are in direct connection with the lymph vessels of nasal mucous membrane. Guye held that aprosexia was owing to the interference with the lymph circulation, by reason of which the products of cerebral tissue metabolism are accumulated in the brain, producing brain fatigue or the so-called 'retention-

[1] Ribot holds that *acts of attention* are accompanied by a temporary suspension of the respiratory rhythm. The air hunger, depending upon the presence of adenoids, therefore of necessity interferes with the psycho-physiology of the act of attention. In this way he would explain the mental state of such children.

exhaustion.' (*d*) It is barely possible that there may be some direct oxidation by the central nervous system, by means of the olfactory bulb (as in some animals), which function, if it exists in man, would be prevented by obstructive lesions of the nose."

Exceptionally a pure reflex case may present itself. In the vast majority, other causes exist, the removal of which yield brilliant results. In view of the marked improvement and entire disappearance of local facial spasms at times, following the relief of the naso-pharyngeal disease, some relation of cause and effect must be admitted. The deleterious effects of the nasal disorder upon the blood and lymph circulation in the brain, and the accompanying anatomical changes, are responsible, in a measure, for the various neuro- and psycho-pathic manifestations occurring in *neurotic* subjects.

Jacobi, in an article published in 1886, directed attention to "partial, and sometimes general, chorea minor from naso-pharyngeal reflex." During the past ten years, the writer has seen quite a number of cases (at the Vanderbilt Clinic) improve under local treatment directed to the naso-pharynx, arsenic being given at the same time, though arsenic and tonics alone failed to make much impression.

Nasal obstructions (inflammatory or otherwise) no doubt act as factors in the production of asthma, in a number of cases — not, however, as the sole agent. A neurosis which remains active after the nasal trouble is relieved is generally found.

In the discussion of this part of our subject, three conditions must be considered : First, predisposition, varying in degree in different individuals ; second, some abnormality or undue excitability of the mucous membrane in some portion of the air passages ; and, finally, a distinct irritant, which, in consequence of individual idiosyncrasy, is reflected to, and again from, the respiratory centre. The greater the predisposition, the less the exciting cause needs to be. The truth of this was frequently exemplified in our experience at the Clinic. In numerous cases, the tendency to bronchitis was relieved by freeing the naso-pharynx. Yet the attacks of asthma would occur, sometimes less frequently ; in other instances, no benefit resulted ; now and then a cure was noted, probably in cases in which the predisposition was slight.

For a moment, attention will be directed to a brief study of the cases in which enuresis is observed. In some the incontinence is nocturnal, in a larger number it is both nocturnal and diurnal. A neurotic condition, with anæmia and flabby muscles generally, is frequently found to be associated with the urinary difficulty. Increased thirst and polyuria add to the distress. As to an explanation, a plausible solution is offered in the mental state incidental to mouth-breathing. Apathy and listlessness, with disturbed intelligence and

deficient innervation in general, are present,—*conditions*, manifestly the result of the obstructed circulation in the blood and lymphatic vessels at the base of the brain and vault of the pharynx.

The higher inhibitory centres, for reasons given, do not act in a normal manner; the bladder reflex, consequently, is not respected, and incontinence follows. Furthermore, the frequent indulgence in water, to relieve the thirst caused by the parched condition of the lips and tongue, produces increased flow of urine, another factor in the etiology. Drugs are of very little service under such circumstances; to cure these patients, the pathological state in the naso-pharynx must be removed.

An attempt has been made to present the more important features. Much might be added. The instructions in the following, taken from the paper of Jacobi, are to the point. If carefully followed, many evils may be avoided and a great deal accomplished in the way of prophylaxis.

Jacobi writes:

"I have always made it a rule to keep all the integuments clean. At least once a day a physiologic solution of salt water is poured through the nares of every infant or child over whom I have control. Big adenoids should be removed, large tonsils resected. There is more danger in a dirty nose than in an unwashed face. Only do not be satisfied with merely ordering it. I have met many a 'trained' nurse who did not know how to inject or to irrigate a nose. A mother or a child's nurse should be instructed by you personally how to do it. Here, as everywhere, when two do the same thing, it is by no means the same. There are many cases of nasal diphtheria, such as are most likely to resist the influence of antitoxin, which are still spared a fatal termination by persistent and correct irrigation of the nares and naso-pharynx."

Pure air and sunlight are indispensable to health. The air should enter the lungs by way of the nasal passages; "And breathed into his nostrils the breath of life," we find recorded in Genesis.

"There is more than a grain of truth in the aphorism, 'Shut your mouth and save your life,' found on the title-page of Captain Catlin's celebrated pamphlet on mouth-breathing."

The homely, forcibly expressed dictum of Catlin must not be lost sight of. Give the little patients free nasal respiration, and give it to them early—the earlier the better.

Preventive medicine has done much to alleviate human suffering. Efforts in this direction have already borne fruit, and as a knowledge of etiology increases, advance in prophylaxis will keep pace.

These assertions are particularly applicable to our subject. To sum up, we may add:

(1) The removal of the lymphoid hypertrophies in the naso- and oro-pharynx, with the cure of the associated naso-pharyngeal catarrh, will restore the patency and permeability of the nose. If done early, many local pathological changes may be avoided.

(2) The general health will be more or less improved.

(3) The mental faculties and general intelligence will be improved.

(4) Defects in speech and in hearing due to nasal troubles will disappear.

(5) Deaf-mutism may be relieved.

(6) The functions of taste and smell will be restored.

(7) Reflex neuroses of various kinds will be modified or cured.

(8) Nasal and supposed pulmonary hemorrhages will disappear.

(9) Thoracic deformities will be relieved or cured.

(10) The tendency to acute rhinitis, pharyngitis, laryngitis, bronchitis, and pneumonia becomes less and less with the restoration of normal respiration.

(11) The dangers attending the presence of enlarged cervical glands will be avoided.

(12) The invasion of various infectious diseases is less likely when the nasal mucous membrane is in a healthy state.

(13) The danger of meningeal infection from the naso-pharynx will be lessened.

(14) Ear complications in general, and particularly those incidental to the infectious diseases, will be avoided or rendered less dangerous.

SCARLATINA MILIARIS.

By J. P. CROZER GRIFFITH, M.D.

THAT an eruption of miliarial vesicles may attend the ordinary rash of scarlatina is well known, and is referred to by very many text-books. Yet considerable contradiction exists between the statements regarding the subject, although the majority appear to agree in certain points which seem to me open to question. It is for this reason that I am led to detail the histories of a few cases, which illustrate certain matters to which attention may well be drawn. What is said refers chiefly not to a very slight or indistinct development of vesicles in scarlatina, which is a symptom of great frequency, but to the great development of the symptom, constituting the *scarlatina miliaris* of older writers.

Both the general trend of opinion and the variance in the statements of writers are illustrated by the following extracts from a few text-books at hand:

Atkinson (article in the *Reference Handbook of the Medical Sciences*) speaks of an eruption of sudamina, which is especially liable to develop in warm weather, or in children too warmly covered with bedclothes. He is in entire lack of accord with Gee (*Reynold's System of Medicine*, 1868, i., 366), who speaks of the occasional widespread development of miliaria, which is, he thinks, not connected with sweating, but rather with the age of the child and the amount of eruptive swelling of the cutis. He associates it with the intensity of the scarlatinal rash. Squire (*Quain's Dictionary of Medicine*) likewise denies any causative influence of perspiration, and states that the vesicles appear where the scarlatinal rash is most intense. Bohn (*Gerhardt's Handbuch der Kinderkrankheiten*, ii., 259) regards the development of miliaria as accompanying only an intense scarlatinal eruption with considerable serous exudate in the skin.

Rilliet and Barthez (Sanné) (*Maladies des Enfants*, 1891, 78, 79, 92, 100) speak of miliaria being rare in young children but common in adults. They associate it with intensity of the scarlatinal rash, and

state that where there have been numerous vesicles the skin becomes detached in large leathery placques. D'Espine and Picot (*Maladies de l' Enfance*, 5th ed., 26) refer to the occurrence of vesicles, due probably to an especially active hyperæmia of the skin. Moizard [*Traité des Maladies de l' Enfance* (Grancher, etc.), i., 124, 125, 129] also associates vesicles with an intense ordinary eruption, and clearly holds the view that an abundant peeling will take place where the vesicles were most abundant. He says that miliaria occurs frequently. On the other hand, Baginsky (*Lehrbuch der Kinderkrankheiten*, 5. Aufl., 126, 128) says that the eruption is seen but seldom and only in severe cases, thus differing from Moizard, as he does from Ashby and Wright (*Diseases of Children*, 2d ed., 241), who regard "sudamina" as not uncommon. Vogel (Biedert) (*Lehrbuch der Kinderkrankheiten*, 10. Aufl., 485) writes of the presence of innumerable vesicles over the surface of the body, occurring generally only in well-developed, severe cases. West (*Diseases of Infancy and Childhood*, 6th ed., 813) says that now and then sudamina are mingled with the eruption. Henoch (*Vorlesungen über Kinderkrankheiten*, 8. Aufl., 641) states that a miliarial eruption may develop in certain areas or over nearly the whole body, but he makes no reference to the relation of this to the intensity of the scarlatinal eruption. He has seen widespread miliary scarlatina in all the members of one family.

There are certain other writers who refer to the matter either not at all, or very briefly and without comment upon its significance. Thus Forchheimer (*Twentieth Century Practice of Medicine*, xiv., 40) merely speaks of *scarlatina miliaris*, characterized by the development of small vesicles; Hatfield (*American Text-Book of the Diseases of Children*, 1st ed., 163) does the same; Guinon [*Traité de Médicine* (Charcot), i., 50] states that vesicles sometimes appear on the neck, waist, and abdomen; Eustace Smith (*Disease in Children*, 2d ed., 36) merely refers to the fact that miliaria may occur; and Holt (*Diseases of Infancy and Childhood*, 892) says only that an eruption of miliaria has been observed. Whittaker [*American Text-Book of Medicine* (Pepper)], Rotch (*Pediatrics*), and J. Lewis Smith (*Diseases of Children*) do not mention the symptom at all.

A review of the points of agreement and of difference of these writers shows that a number either have not observed miliaria in scarlet fever, or have not thought its presence deserving of mention. The majority, however, refer to it, although in different ways. The view that the eruption has anything to do with perspiration is not held by most. The greater number clearly believe that the vesicles develop only when the ordinary scarlatinal eruption is very intense. Some

make it a symptom of an unusual degree of serous infiltration of the skin. Some believe that it is seen in severe cases only. Some claim that it is followed by an abundant peeling desquamation.

One opinion I have still reserved, partly because of the importance which its source gives it, partly because it is at variance with most, and, in some respects, with all, of those quoted. I refer to that of Thomas (*Ziemssen's Cyclopædia of Practical Medicine*, Eng. trans., ii., 208). This writer states that a miliarial eruption, *scarlatina miliaris*, is sometimes seen, and may appear in all parts of the body. In some epidemics it has been noted so often, and in such abundance, that the normal eruption was observed only in the minority of cases. He regards its presence as depending solely on a peculiar disposition of the skin of the patients. Usually only a few vesicles can be found.

The following are the case-histories I wish to detail:

Case I. — Philip Smith, six years old, entered the Children's Hospital, May 2, 1899, suffering with malarial fever. Under the influence of quinine he recovered promptly. On the 14th his temperature rose; he was chilly, and soon developed a scarlatinal rash, with sore throat and headache. On the 17th, a miliarial eruption was present in abundance on the neck and head. On the 18th, there was slight branny desquamation. On June 3d, typical scarlatinal peeling appeared on the hands and legs.

This case is an ordinary one of miliarial eruption attending scarlet fever. It is given here merely as an illustrative example. The redness of the scarlatinal efflorescence was decided, the branny desquamation appeared early on the seat of the miliaria, and no peeling was seen until much later. It is to be observed, however, that the rash was no more intense than in hundreds of other cases which do not exhibit miliaria; that the attack was not at all a severe one; and that the characteristic peeling appeared not in regions where there had been miliaria, but elsewhere.

Case II. — Elmer Pritchett, seven years old. *May 11, 1897:* On the 9th the father noticed a red, itching vesicular eruption, diffuse over the body, most marked on the back and abdomen. The child seemed well otherwise, the appetite good, the bowels regular. The father thinks that there has been no fever. *Examination:* Over the trunk and extremities there is a most extensive development of small vesicles, closely crowded on a base which, on closer inspection, appears to be red and punctate, resembling the rash of scarlet fever. The temperature is 101°; the pulse 120. The throat is normal in appearance. *May 12th:* The rash on the hands is typically vesicular, with but little redness. The temperature is 99.+8°. The case appears to be one of unusually extensive miliaria. *May 14th:* The child has been sent to Dr. Arthur Van Harlingen for an opinion, who confirms the diagnosis of miliaria, and says that he has but once before seen so diffuse a case. The condition is now improving, and the rash drying. *Later in the month:* The skin is now scaling profusely in a manner entirely characteristic of scarlatina. Over the nates the dried vesicles form almost crusts of desquamating skin.

I believe that I erred entirely in the diagnosis of this case, and that

it was undoubtedly scarlatina with unusually abundant miliaria. It is to be noted that the innumerable vesicles obscured the character of the scarlatinal eruption beneath; that this latter eruption was not more intense than in an average case, and in some places, as on the hands, much less red than usual; that the whole attack was clearly a very mild one, as the boy did not feel ill, there was very little fever, and no involvement of the throat.

Case III.—Katie Carey, four years old. *May 27, 1897:* On the night of the 25th, she had fever, vomiting, and a croupy cough, and on the morning of the 26th, is said to have been covered with a red rash. To-day it is said to have faded slightly. *Examination:* Over the body there is a rather indistinct rash resembling that of scarlatina, while on the hands and arms there is also an extensive vesicular eruption. The temperature is 101° (P.M.); the tongue bright red over the anterior portion; the tonsils rather red and swollen. *May 28th:* The temperature is 100.8° (P.M.); the tongue bright red; there is no glandular involvement; the throat is only slightly red; there are no spots on the tonsils; the general condition is excellent. The rash has now a more characteristically scarlatinal appearance; yet about the waist and nates it is very much mottled like a fading eruption of measles, with slight pigmentation of the skin. Then, too, the hands are covered so thickly with minute vesicles on a red base, that the condition presents a strong resemblance to the dermatitis of rhus poisoning.

This case shows a good development of miliaria without this seeming to depend in the slightest on the intensity of the scarlatinal eruption. The diagnosis on the 27th was most uncertain.

Case IV. — Frank Matteaci, three years old, entered the Children's Hospital, March 22, 1899, suffering with an attack of typhoid fever. The typhoid roseola was present. His temperature ranged from 101° to 103° until the 27th, when it began to grow less. From March 30th, to April 1st, the temperature was 100° or less. On the morning of April 3d, a rash appeared over the body, especially marked on the face, trunk, and thighs. It was typically scarlatinal, but not very red, and there was in addition an almost universal development of closely crowded minute vesicles which made the whole resemble an aggravated attack of miliaria. The throat was sore and red, with a white patch on the tonsil; the tongue was red. On April 5th, the rash was still red, but the vesicles were beginning to dry, leaving the skin rough. The vesicles were especially marked on the forehead, chest, and abdomen. On April 8th, a profuse branny desquamation was present, but no peeling. On April 11th, this desquamation was still present in abundance, with very slight peeling on the abdomen and back; so slight that it could have been overlooked easily. The temperature range of the attack was as follows:

April 2,	A.M.,	97.°	P.M.,	99.6°	April 5,	A.M., 99.°	P.M., 99.2°
" 3,	"	99.8	"	99.8	" 6,	" 100.2	" 100.
" 4,	"	100.	"	99.	" 7,	" 98.8	" 99.

With such a temperature range as the above, and with an eruption as varying from the ordinary type as this, it is safe to say that the diagnosis of scarlatina would hardly have been made by us had there not been an epidemic of the disease prevalent in the house at this time. The case is interesting apart from the almost afebrile condition and great mildness of the attack, both because the development of

vesicles was out of all proportion to the intensity of the redness of the skin, and because the abundant miliarial eruption was without any extensive characteristic peeling as in the other three cases.

It is impossible to use but a few cases as positive evidence in drawing conclusions; but for negative evidence even a single case is valuable, and may disprove statements which are made in the form of generalizations. That is to say, while it may take a great number of instances of association of two symptoms in a certain disease to prove that they bear some relation to each other, a very few cases of the occurrence of one of these symptoms in a well-marked state, while the other is entirely absent, compel the suspicion that they are in no way related. Consequently the cases I have reported suggest the following thoughts:

1. However frequently miliaria may attend severe cases of scarlatina, the oft-expressed view seems untenable, that its presence is an indication that the case is severe. In none of these four cases was the attack severe, and it was lighter in the two (Cases II. and IV.) with the greatest development of vesicles.

2. The same statement applies to the intensity of the scarlatinal eruption and its relation to miliaria. There appears to be no connection between the two. In Case IV., with an enormous number of miliarial vesicles, the scarlatinal redness was poorly developed. There seem to be different causes acting to produce the efflorescence and the vesiculation, although these causes are undoubtedly frequently associated.

3. Contrary to the opinion of certain writers there appears to be no necessary relation between the amount of scarlatinal peeling and the degree of miliarial eruption. In Case IV., with numerous vesicles, peeling was scarcely discoverable, while in Case II., with an equal development of miliaria, peeling was abundant.

4. The view of Thomas is very probably correct (and, in so far as they differ, those of the other writers quoted are incorrect), viz.: that the development of miliaria in scarlatina depends largely on some peculiarity in the skin of the patients, rather than on any special intensity of the scarlatinal rash or other factor. The observation of Henoch upon a family tendency to miliary scarlet fever seems to support this view.

5. It is perfectly possible in occasional cases to have the presence of abundant miliarial eruption cause decided difficulty in diagnosis, and even lead into error. In Case II. a mistake was made in spite of careful examination, and in Case IV. the fact of the existence of a house epidemic was the deciding factor in determining the diagnosis.

UBER TONSILLITIS CHRONICA LEPTOTHRICIA BEI KINDERN.

VON DR. ALOIS EPSTEIN,

PROFESSOR DER KINDERHEILKUNDE AN DER DEUTSCHEN UNIVERSITÄT PRAG.

IM Jahre 1873 machte B. Fraenkel[1] auf eine bei einem Erwachsenen beobachtete Affection des Rachens und des Zungengrundes aufmerksam, welche er als Pharyngomycosis benigna bezeichnete. Aus der Beschreibung sei hervorgehoben, dass auf der Schleimhaut des Rachens, besonders an den Mandeln, weisse erhabene Flecken vorhanden waren, welche der Unterlage fest anhafteten und, mikroskopisch untersucht, aus Epithelien und massenhaften Pilzen u. z. zumeist aus *Leptothrixformen* bestanden. Seither haben auch andere diese Krankheit beobachtet und unter demselben Namen oder als Pharyngomycosis leptothricia (Heryng[2]), Mycosis tonsillaris benigna (E. Fraenkel[3]), Algosis faucium leptothricia (Jacobson[4]) beschrieben. Der letztgenannte Autor hat im Jahre 1888 die bis zu dieser Zeit veröffentlichten 17 Fälle dieser Art gesammelt und einige Fälle eigener Beobachtung hinzugefügt. Seither ist eine weitere Reihe von einschlägigen Beobachtungen über diese eigenartige Erkrankung des Rachens sowie auch über andere Localisationen der Leptothrix (Rachentonsille, Tubenwülste, Kehlkopf, Bronchien, Lungen) bekannt geworden. Immerhin wird noch die Erkrankung von den Autoren als eine seltene erklärt. Wie ein neuer Bearbeiter dieses Kapitels, F. Kraus,[5] berichtet, hat die Zahl der mitgetheilten Fälle "noch kein halbes Hundert" erreicht. Für die Seltenheit der Erkrankung dürfte auch der Umstand sprechen, dass ich selbst, der alljährlich etwa 1500 erwachsene Personen weiblichen Geschlechtes zu untersuchen und ihren Rachen mit mög-

[1] Fraenkel, B. *Berliner klin. Woch.*, 1873, S. 94, und *Real-Encyklopaedie der ges. Heilkunde*, von Eulenburg, II. Aufl., S. 509, "Pharynx."

[2] Heryng. *Zeitsch. f. klin. Medizin*, Bd. vii.

[3] Fraenkel, E. *Zeitschr. f. klin. Medizin*, Bd. iv.

[4] Jacobson. *Volkmann's Sammlung klinischer Vorträge*, No. 317.

[5] Kraus, F. "Die Erkrankungen der Mundhöhle und der Speiseröhre." *Handb. d. spec. Path. u. Ther. von Nothnagel*, xvi. Bd., i. Th., i. Abth.

lichster Genauigkeit zu besichtigen hat, noch keinen Fall dieser Art bei einem *Erwachsenen* vorgefunden habe.

Die Leptothrix buccalis zeichnet sich durch ihr Wachsthum in langen, ungegliederten Fäden aus, die im mikroskopischen Präparate entweder einzeln oder miteinander verwirrt erscheinen oder, was besonders charakteristisch ist und wie dies bei breiter, fleckenförmiger Ansiedlung auf der Tonsillarschleimhaut in gut gelungenen Präparaten beobachtet werden kann, in paralleler Anordnung zu Bündeln oder garbenförmigen Gebilden vereint sind. Die mykologische Stellung des Pilzes ist derzeit noch nicht genügend geklärt, woran die Schwierigkeit seiner Reincultur auf künstlichen Nährböden Schuld trägt. Auch uns ist dieselbe nicht gelungen, im Gegensatz zu einem anderen Fadenpilz der Mundhöhle, dem Soor, dessen Wachsthum in charakteristischer Verzweigung längs der Gelatine-Stichcultur leicht durchzuführen war. Gegenüber Miller,[1] welcher meint, dass die verschiedensten Mundbakterien die Eigenschaft haben zu Fäden auszuwachsen und deshalb nicht geneigt ist, der Leptothrix eine besondere Stellung einzuräumen, anerkennt die Mehrzahl der Bakteriologien die Leptothrix als eine besondere Art von Spaltpilzen. Die früher als specifisch angenommene Blaufärbung durch Iod-Iodkali hat an Verwertbarkeit für die Diagnose verloren, seitdem man weiss, dass einerseits auch andere Mundbakterien diese tinctorielle Eigenschaft besitzen, andererseits aber bei sicherer Leptothrix nicht immer Blaufärbung, sondern Braunfärbung eintritt. Ich selbst habe nach Iodzusatz in der Regel die letztere und nur ausnahmsweise eine schwache Blaufärbung gesehen.

Auch die Ansichten über die pathologische Bedeutung der Leptothrix sind widersprechend. Manche Autoren sehen den Pilz als einen gemeinen Saprophyten der Mundhöhle an, dem selbst bei pathologischen Processen nur eine secundäre Rolle zukomme, oder sie heben hervor, dass er an und für sich keine localen oder allgemeinen Krankheitserscheinungen hervorrufe und dass diese letzteren, wo sie auftreten, einer Mischinfection zuzuschreiben sind. Dagegen betonen andere Autoren, insbesondere die klinischen Beobachter der Pharyngomycose mehr oder weniger die krankheitserregenden Eigenschaften der Leptothrix und sprechen mit Rücksicht auf die dabei manchmal zu beobachtenden Beschwerden geradezu von einer *Angina leptothricia*. Eine Stütze für diese letztere Ansicht haben auch einzelne pathologisch-anatomischen Befunde geliefert. Hierher gehört unter anderen ein von Dubler[2] beobachteter Fall eines 8 Monate alten Kindes, bei welchem die Section eine über den Pharynx, Larynx, und Oesophagus

[1] Miller. *Die Mikroorganismen der Mundhöhle*, II. Aufl., 1892, S. 386.
[2] Dubler. *Virchow's Archiv*, cxxvi. Bd., S. 454.

verbreitete, mit Epitheldefecten und entzündlichen Erscheinungen einhergehende Leptothrixmycose ergab. Mir ist diese Beobachtung um so interessanter, als sie mich an mehrere aus meiner Klinik stammende Fälle älteren Datums erinnert hat, welche Kinder der ersten Lebensmonate betrafen und bei welchen die Section (Prof. Klebs, Prof. Eppinger) eigenthümliche Veränderungen der Oesophagusschleimhaut constatierte (festhaftende Beläge, grosse inselförmige Epitheldefecte mit gerötheten Rändern, Erweichung des Gewebes) und der Process als durch Leptothrix verursacht, die sich in ausgebreiteten und dichten, das Epithel durchwuchernden Rasen vorfand, aufgefasst wurde.

Bei dieser Unsicherheit der Stellung dieses Pilzes in der Pathologie erscheinen deshalb weitere klinische Beobachtungen über einschlägige Affectionen wohl berechtigt, insbesondere auch deshalb, weil sie *Kinder* betreffen.

Bezüglich des Alters der mit Leptothrixmycose des Rachens behafteten Kranken, deren Krankengeschichten unter diesem oder ähnlichen Titeln mitgetheilt wurden, sei erwähnt, dass dieselben erwachsene Individuen betrafen, so dass Boulay[1] (1895) daraufhin den Satz ausspricht: "C'est une maladie de l'âge adulte." Die Beobachtungen an Kindern sind allerdings so spärlich, dass dieselben der Beachtung wohl entgehen konnten. Als eine der wenigen und ersten möchte ich eine Bemerkung von Unterholzner[2] (1885) erwähnen, welcher in einer vorwiegend statistischen Arbeit über Diphtheritis kurz angibt, dass er bei 3 Kindern (einem 15-jährigen Knaben, einem 10-jährigen Mädchen und bei "einem kleineren Kinde") eine Mycosis an den Tonsillen beobachtet habe. Es verdient hervorgehoben zu werden, dass bei einem dieser Fälle, welcher mit Fieber und Drüsenschwellung einherging, im ersten Augenblicke an Diphtheritis gedacht wurde, bis die mikroskopische Untersuchung den sicheren Aufschluss gab, indem neben Epithelien Leptothrixfäden nachgewiesen wurden und auch der weitere Verlauf gegen die erste Annahme sprach. Ausser dieser unbeachtet gebliebenen Mittheilung Unterholzner's und der schon erwähnten Beobachtung von Dubler findet sich in einer Arbeit von Stooss,[3] welche sich mit bakteriologischen Untersuchungen der Anginen bei Kindern befasst, einige Fälle von "Anginen mit vorwiegend Leptothrix" verzeichnet; doch möchte ich nur *einen* Fall (No. 5; 12-jähriges Mädchen, dessen Mutter an derselben Affection litt) als hierher gehörig betrachten. Bemerkenswert ist ferner ein von R. Fischl[4] beobachteter Fall eines

[1] Boulay. *Manuel de Médecine*, T. v., p. 159, Paris, 1895. Rueff et Cie.
[2] Unterholzner. *Jahrb. f. Kinderheilkunde*, xxiii. Bd., S. 245.
[3] Stooss. *Zur Aetiologie u. Pathologie der Anginen, etc.*, 1895. Carl Sallmann.
[4] Fischl, R. *Prager med. Wochenschr.*, 1899.

3-jährigen Mädchens, bei welchem nach spontaner Ausheilung des Processes "ausgebreitete, an luetische Zerstörungen erinnernde Defecte im Bereiche der Gaumenbögen und des Zäpfchens" zurückblieben. Zu erwähnen ist ferner ein ziemlich allgemein gehaltener Aufsatz von Lubet-Barbon,[1] aus welchem jedoch nicht zu entnehmen ist, ob demselben eigene Beobachtungen zu Grunde liegen. Mit wenigen Worten erwähnt endlich Cuvillier[2] in seiner Abhandlung über die chronische Pharyngitis der Kinder auch die Pharyngomycose leptothrixique.

Wenn ich auch nicht verbürgen will, dass mit diesen Angaben die pädiatrische Litteratur erschöpft ist, so ist doch jedenfalls festzustellen, dass diese zerstreuten Mittheilungen einen nachhaltigen Eindruck nicht hinterlassen haben. Die zahlreichen Lehrbücher der Kinderkrankheiten bewahren über die genannte Affection der Tonsillen ein consequentes Stillschweigen, wiewohl manche Autoren bei der Besprechung der Differentialdiagnose der verschiedenen Beläge an den Tonsillen, selbst Schleimgerinnsel, Speisereste, Kalkablagerungen u. s. w. als Umstände anführen, welche einen Belag vortäuschen können. Da aber gerade Kinderärzte gewohnt sind die Rachengebilde genau zu besichtigen, so lässt sich daraus schliessen, dass die Angina leptothricia zu den selteneren Rachenaffectionen der Kinder gehört. Trotz dieser verhältnismässigen Seltenheit verdient sie jedoch, weil sie gerade in diesem Alter um so eher zu diagnostischen Verwechslungen Anlass geben kann, schon aus diesem Grunde die vollste Berücksichtigung.

Meine Beobachtungen betreffen 5 Fälle dieser Art, welche sämmtlich meiner Privatpraxis entstammen, wogegen ich über keinen Fall verfüge, der in meiner Klinik oder Poliklinik beobachtet worden wäre. Der Grund dieses Verhaltens dürfte wohl darin zu finden sein, dass der Verlauf dieser Halsaffection ein chronischer ist und ihr Charakter gewöhnlich erst im Laufe längerer Beobachtung erkannt wird. Sämmtliche dieser Kinder standen seit ihrer frühesten Lebenszeit unter meiner ärztlichen Beobachtung. Die beiden ersten Fälle betrafen ein Geschwisterpaar. Der Vater litt an chronischer Lungenphthise, an welcher er später starb. Die Mutter gehört zu jenen Frauen, welche den Hals ihrer Kinder oft besichtigen und sich dadurch eine gewisse Fertigkeit im Erkennen krankhafter Veränderungen aneignen.

Fall 1. Acht Jahre altes Mädchen. In seinem zweiten Lebensjahre wurde dasselbe an schwerer Diphtheritis und später einigemale an follicülärer Angina von mir behandelt. Ich sah das Kind oft und untersuchte den Rachen desselben bei verschiedenen Anlässen. Im Mai 1893, zu dem Kinde gerufen, fand ich an beiden, mässig hypertrophierten Tonsillen

[1] Lubet-Barbon. *Annales de Médecine et Chirurgie infantile*, T. ii., p. 721, 1898.

[2] Cuvillier. *Traité de maladies de l'enfance*, T. ii., p. 443, Paris, 1897. Masson et Cie.

einzelne stecknadelkopfgrosse, mattweisse, rundlichzackige Auflagerungen. Dabei Röthung der Schleimhaut, Schlingbeschwerden und Fieber. Die letzteren Erscheinungen schwanden nach 3–4 Tagen. Der lokale Befund blieb jedoch unverändert. Im Laufe der Beobachtung nahmen einzelne Flecken an Umfang zu und traten noch andere auf. Das subjective Befinden war ein gutes, nur an manchen Tagen klagte das Kind über mässige Schmerzen beim Schlingen. Die Behandlung mit verschiedenen Gurgelwässern blieb ohne Erfolg. Nach dreiwöchentlicher Dauer, während welcher das Mädchen von seinem jüngeren Bruder isoliert blieb, konnte die Meinung, dass es sich um eine gewöhnliche Tonsillitis handle, nicht mehr aufrecht erhalten werden. Um so räthselhafter wurde die Diagnose. Die hereditäre Belastung und das durch längere Zeit bestehende schlechte Aussehen des Kindes liessen an die Möglichkeit einer Tuberculose der Tonsillen denken, um so mehr als an einzelnen Stellen auch kleine, flache Erosionen des Epithels auftraten. Dies veranlasste mich zu einer Untersuchung der Auflagerungen, von denen mittels Pincette kleine Partikel abgehoben wurden. Die Untersuchung auf Tuberkelbacillen war negativ. Dagegen zeigte die mikroskopische Besichtigung ein Bild, welches ich bis dahin am Lebenden nicht gekannt hatte. Die von verschiedenen Stellen und wiederholt entnommenen Präparate bestanden neben spärlichen, abgestorbenen Plattenepithelien aus langgestreckten, unverzweigten, parallel verlaufenden, zu dichten Büscheln angeordneten Fäden. Nach dieser Aufklärung behob ich die Isolierung und liess das Kind wieder zur Schule gehen. Ende Juni reiste die Familie in einen Curort ab, welcher dem Vater seines Leidens wegen empfohlen worden war. Von dort schrieb die Mutter, dass die Flecken an den Mandeln noch vorhanden seien. Mitte November sah ich das Kind wieder. Die Affection bestand weiter und hatte nur ihre äussere Form verändert. Die Mandeln waren inzwischen etwas grösser geworden. Im Laufe der nächsten Monate hatte ich oft Gelegenheit, das Kind zu untersuchen. Die subjectiven Beschwerden bestanden nur in von Zeit zu Zeit auftretenden Halsschmerzen. Unter allmäliger Abnahme schwanden die Auflagerungen erst Ende Juni 1894, also nach mehr als einjährigem Bestande und haben sich auch nicht mehr erneut. Die Tonsillen haben sich später bis zur normalen Grösse verkleinert.

Fall 2. Fünf Jahre alter Knabe, schwächlich, blass, Nackendrüsen geschwellt. Im December 1893, während beim vorigen Kinde die Affection der Tonsillen in vollster Blüte stand, wurde ich von der Mutter aufmerksam gemacht, dass sich auch bei dem jüngeren Bruder ähnliche Flecken an den Mandeln zu bilden beginnen. Die von Zeit zu Zeit vorgenommene Inspection und die wiederholte mikroskopische Untersuchung, sowie auch der weitere Verlauf zeigten, dass es sich um die gleiche Krankheit handle. Die hypertrophischen Mandeln bedeckten sich mit isolierten, manchmal auch confluierenden mattweissen Auflagerungen, die allmälig an Zahl und Grösse zunahmen und im Lauf der Zeit ein sehr wechselndes Aussehen darboten. Der Knabe klagte öfters über Halsschmerzen und Schlingbeschwerden. Da diese Beschwerden gewöhnlich nur kurz anhielten, dafür aber sich oft wiederholten, der Knabe sonst ganz munter, fieberfrei und bei Esslust blieb, so war für die Mutter und ebenso für mich die Frage des Ausgehens oder Zuhausebleibens an der Tagesordnung. Auch bei diesem Kinde beobachtete ich zuweilen zwischen den Auflagerungen die Entwicklung ganz oberflächlicher bis linsengrosser Erosionen an den Tonsillen, den Gaumenbögen oder den seitlichen Wänden der hinteren Rachenwand. Die Affection bestand durch nahezu 1½ Jahre. Die Auflagerungen schwanden in der Weise, dass sie sich an einzelnen Stellen in länger ausgezogene, wie gestielte, gelbliche, verhornte Prominenzen oder kurze Stacheln verwandelten, welche schliesslich abgestossen wurden.

Fall 3. Zehn Jahre altes Mädchen, neuropathisch belastet. Beide Mandeln vergrössert, von blätterigem Bau und mit tiefen Lacunen versehen. An der hinteren Rachenwand adenoide Vegetationen. Das Mädchen klagt oft über Halsschmerzen. Als Ursache derselben konnte öfters eine folliculäre Tonsillitis nachgewiesen werden. Die Schmerzen traten aber auch sonst häufig auf, ohne dass eine nachweisbare Veränderung an den Rachengebilden zu constatieren war. Die häufigen und andauernden Klagen legten schon den Verdacht einer

Simulation nahe. Bei einer im Mai 1894, aus demselben Grunde vorgenommenen Untersuchung des Rachens entdeckte ich hinter dem oberen Pole der rechten Mandel in dem durch den Zusammenfluss der beiden Gaumenbögen gebildeten oberen Winkel der Mandelnische einen mehr als erbsengrossen, glatten, etwas plattgedrückten, hellweiss glänzenden Körper von käsiger Consistenz. Derselbe war gewöhnlich durch den oberen Pol der vergrösserten Tonsille verdeckt und wurde nur bei einer bestimmten Stellung des Gaumensegels oder wenn die Tonsille mit dem Spatel oder einem stumpfen Haken nach abwärts gedrängt wurde, gut sichtbar. Die mikroskopische Untersuchung der Masse ergab neben verhornten Epithelien hauptsächlich ein dichtes Filzwerk von langen unverzweigten Fäden. Nach mehrere Monate langem Bestande, auf welchen auch die örtliche Behandlung mit Iodtinktur u. s. w. keinen Einfluss übte, wurde im November 1894, die Auflagerung, soweit als sichtbar, mit stumpfer Pincette entfernt. Im Februar 1895, traten neuerdings öfters sich wiederholende Halsschmerzen auf. Die Untersuchung ergab, dass die Auflagerung u. z. an derselben Stelle sich wieder erneut hatte. Ausserdem lugte auch auf der correspondierenden Gegenstelle hinter dem oberen Pole der linken Mandel ein ähnliches Gebilde hervor. Der Zustand blieb durch einige Wochen stationär, die Herde schienen nur ihre Grösse zu verändern. Schliesslich entfernte ich mit Löffel und Pincette unter geringer Blutung die ziemlich festhaftenden Massen. Die mikroskopische Untersuchung ergab denselben Befund wie früher. Die Affection bestand durch fast ein Jahr.

Fall 4. Achtjähriges Mädchen. Dasselbe beklagt sich angeblich seit Wochen über zeitweilig auftretende Halsschmerzen, denen man jedoch, da Fieber fehlte, keine weitere Bedeutung beilegte. Manchmal ist die Stimme belegt. Im April 1898, zur Untersuchung aufgefordert, fand ich an beiden Mandeln buckelförmige, mattweisse Erhabenheiten, deren Form mich schon die Art der Erkrankung vermuthen liess. Die mikroskopische Untersuchung bestätigte die Diagnose. Das Kind ist sehr blass. Die Drüsen am Halse sind bis Haselnussgrösse geschwellt. Nach zweimonatlichem Bestande und Gurgelungen mit Haller Iodwasser, dreimal täglich, schwanden die Belege und Drüsenschwellungen, das Aussehen besserte sich wesentlich.

Fall 5. Siebenjähriger Knabe. Mit 1 und 2 verwandt und in demselben Hause wohnend. Ich entdeckte die Affection, die keine Beschwerden zu verursachen schien, ganz zufällig, als ich im September 1899, einen älteren Bruder wegen einer acuten exsudativen Tonsillitis zu behandeln hatte. Ausser den Flecken an den Tonsillen fanden sich auch die Papillæ circumvallatæ des Zungengrundes stark vorspringend und wie mit einem dichten grauen Schleier überzogen. Die Affection erhielt sich unverändert durch mehr als zwei Monate. In letzter Zeit (Mitte December) haben sich unter Iodwasser-Gurgelungen die Mandeln gereinigt, während einzelne Papillen des Zungengrundes noch immer mit dem grauen Leptothrix-Belage bedeckt sind und sich durch diesen letzteren von jenen Papillen deutlich unterscheiden, an denen sich der Belag schon abgestossen hat und welche stark geschwellt und dunkel geröthet vorragen.

Wie an den hier mitgetheilten und besonders den drei ersten Fällen ersichtlich ist, ist die Krankheit durch einige charakteristische Eigenschaften ausgezeichnet, welche sie von den übrigen, mit einem Belage einhergehenden Erkrankungen der Tonsillen wohl unterscheiden. In dieser Beziehung sind insbesondere hervorzuheben: Die klinischen Erscheinungen, der chronische Verlauf, die Hartnäckigkeit, mit welcher sie therapeutischen Eingriffen widersteht und der mikroskopische Befund.

Durch die fortlaufende Beobachtung einzelner Fälle war ich in der Lage, die Eigenthümlichkeiten und Stadien der Affection schärfer zu

verfolgen. Anfangs sieht man rundliche oder rundlichzackige stecknadelkopfgrosse, bis zu Linsengrösse sich vergrössernde, nur wenig erhabene Flecken von mattweisser, milchglasartiger Farbe zerstreut über den Tonsillen. Das Epithel streicht anfangs noch unverletzt über dieselben. In diesem Stadium ist die Entnahme eines Präparates noch recht schwierig, gelingt kaum ohne kleine Blutung, die Masse verhornten Epithels überwiegt, die Leptothrixfäden sind vielfach verletzt und in unregelmässiger Anordnung. Später treten die Auflagerungen stärker hervor, erscheinen mehr gewölbt oder als deutlich hervortretende Knötchen oder bilden durch Confluenz plaqueartige Formen oder streifenförmige Figuren. In einem späteren Stadium, offenbar nach Durchbruch des Epithels, bilden sich kleine troddel- oder fransenförmige Excrescenzen von weisser Farbe und bröckliger Consistenz, welche vorsichtig entnommen, viel besser die büschel- oder garbenförmige Anordnung der Fäden erkennen lassen. Manche exfolieren sich und hinterlassen kleine, scharfumrandete Erosionen der Schleimhaut. Ein noch späteres Stadium, welches in Fall 2 zu beobachten war, ist die Bildung von dünnen, stachelförmigen oder gestielten Fortsätzen. Hier überwiegt, wie schon aus der Consistenz derselben zu schliessen ist, die Verhornung des Epithels. Siebenmann[1] hat aus der Untersuchung solcher aus den Krypten hervorragender "Stacheln" den Schluss gezogen, dass die mächtige Verhornung des Epithels das Wesentliche des Processes sei. Er bezeichnet denselben deshalb als Hyperkeratosis lacunaris und hält die Leptothrixansiedelung als eine secundäre, saprophytische Erscheinung. Mit dieser Auffassung stimmt allerdings das klinische Bild der von mir beobachteten Fälle nicht überein. Der Epithelüberzug der Krypten erschien weder verdickt noch auffallend getrübt, sondern schien von normaler Farbe und zarter Beschaffenheit. Die Krypten waren auch nicht von den Auflagerungen bevorzugt, sondern die letzteren sassen vorwiegend auf der freien Oberfläche der Wülste und schliesslich war in manchen Präparaten die Masse und regelmässige Anordnung der Leptothrixfäden so hervorstechend und das Verhalten des Epithels so untergeordnet, dass man die Pilzansiedelung als die pathogene Ursache des Processes ansehen konnte. In diesem Sinne sprechen auch die Impfversuche von Deckert und Seifert,[2] die durch Übertragung der Pilzmasse von einer Hyperkeratosis lacunaris eine charakteristische Leptothrixmykose erzeugten, welche aber nach wenigen Wochen ausheilte.

Die subjectiven Beschwerden scheinen bei Kindern grösser zu sein, als dies von jenen Autoren, welche nur Erwachsene beobachteten,

[1] Siebenmann. *Archiv f. Laryngologie*, Bd. ii., S. 365.

[2] Deckert und Seifert. *Phys. med. Gesellschaft*, Würzburg, 1880.

angegeben wird. In meinen Fällen klagten die Kinder oft über ein Gefühl von Wundsein, Brennen oder Schmerzen beim Schlingen, waren aber zu Zeiten wieder schmerzfrei. Im Falle 3 stellte sich zeitweise ein hartnäckiges Räuspern und Hüsteln ein. Dies alles schwand vollends, nachdem die Affection gänzlich ausgeheilt war. Auch vorühergehende Fieberbewegungen wurden bei den Kindern manchmal beobachtet. Die Tonsillen waren bei allen Kindern grösser als de norma, doch nicht übermässig gross. Zwischendurch treten ziemlich häufig auch acute Entzündungen mit eitrigem Belag auf. Es scheint, dass die Pharyngomykose durch die mechanischen Störungen, welche sie in den Geweben hervorruft, an und für sich zeitweilige Beschwerden verursacht, andererseits aber auch für Infectionen anderer Art den Boden vorbereitet.

Ein Irrthum der Diagnose und eine Verwechslung mit einer acuten Form der Angina kann recht wohl Platz greifen, wenn man zu einem unbekannten Kinde gerufen wird und bei demselben zufällig locale Reizerscheinungen oder Fieber vorfindet. Die Verlegenheit wächst, wenn sich an dem localen Befunde durch längere Zeit nichts ändern will. Fall 1 demonstriert ein solches Verhalten. Lubet-Barbon erwähnt eines Falles von confluierender Pharynxmykose, der mit Diphtheritis verwechselt und mit Serum injiciert wurde. Im Allgemeinen ist jedoch das Bild dieser Erkrankung genug charakteristisch. Die Leptothrix-Auflagerungen sind gewöhnlich in grösseren Abständen über die ganze Tonsille zerstreut, innig festhaftend, die Umgebung gewöhnlich frei von entzündlichen Erscheinungen. Durch ihre mehr matte Farbe, das trockene Aussehen und die manchmal villöse Beschaffenheit unterscheiden sie sich von den mehr gelblichen oder grauen, zähflüssigen, leichter abstreifbaren Exsudaten und den bei reichlicherem Faserstoffgehalte mehr membranös erscheinenden Auflagerungen der acuten Tonsillitis. Eine Verwechslung mit den vereinzelten safrangelb durchscheinenden Kalkablagerungen oder mit den sehnig gestreiften, die Lacunen brückenförmig übersetzenden Flecken mancher Tonsillen wird kaum möglich sein. Auch die in hypertrophischen Mandeln sitzenden Tonsillarpfröpfe, welche ebenfalls vereinzelte Leptothrixfäden zu enthalten pflegen, unterscheiden sich durch ihren Sitz in den Lacunen, die mehr breiige Consistenz, den Geruch und ihre Ausdrückbarkeit von der hier besprochenen Mykose.

Der chronische Verlauf wird durch therapeutische Eingriffe, namentlich durch Pinselungen, die man schliesslich der Umständlichkeit und langen Dauer wegen der Umgebung zu überlassen pflegt, wenig beeinflusst, bis endlich die Affection von selbst schwindet. Zu den empfohlenen Aetzungen mit dem Thermocauter oder mit Chromsäure oder

zur Abtragung der Mandeln oder Auslöffelung der Herde habe ich nicht gegriffen. Nur im Falle 3, bei welchem die Affection mehr localisiert war, habe ich die Herde mit Löffel und Pincette entfernt. Empfohlen wurden auch Pinselungen mit Nicotin (2:1000) mit Rücksicht auf die Beobachtung, dass Raucher von der Affection verschont bleiben. Ich verwendete Pinselungen mit Alcohol, Chloroformwasser, Iodtinctur oder liess mit Haller Iodwasser (dreimal täglich) gurgeln. Letzteres schien sich am besten zu bewähren.

THE USE OF THE DIPHTHERIA ANTITOXIC GLOBULINS OF THE BLOOD SERUM INSTEAD OF THE ENTIRE SERUM IN DIPHTHERIA.

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EVER since the discovery of the value of diphtheria antitoxin in the prevention and treatment of diphtheria, it has been the desire of those using and producing the serum to separate the diphtheria antitoxin from the blood serum, with the hope that in this way the antitoxic effect might be retained, while the deleterious effects sometimes produced by injections of blood serum, as evidenced in the rashes and the effect on the red blood cells, might be avoided. As investigation has progressed, it has become more and more evident that the antitoxic substances in the blood are closely combined with the globulins of the blood, and that whatever precipitates them precipitates the antitoxin also. In fact, without globulin there appears to be no antitoxin, and wherever antitoxin exists globulin does also. Up to the present time, no difference has been detected between globulin which is not antitoxic and that which is, except in the specific characteristic of neutralizing toxin. The latest investigations on this subject have been made by Atkinson[1] in the Research Laboratory of the Health Department of the City of New York and by Seng[2] in Germany.

At the Research Laboratory of the Department of Health of the City of New York, we have made it a point to draw blood from all the horses which are to be used for the development of antitoxin before they have received any injections of toxin. This so-called normal blood as well as the later drawings of antitoxic blood are tested for the amount of globulin and the amount of antitoxin.

A most remarkable relation between the increase of antitoxin and the increase of globulin has thus been shown. These tests have been made by Mr. Atkinson, the Assistant Chemist, and through his kindness I quote the following recent tests of a number of blood drawings

[1] *Journal Experimental Medicine*, November, 1899.

[2] *Zeitschrift für Hygiene und Infectionskrank.*, Bd. xxxi, p. 513 (1899).

from a horse (No. 137) showing rapid variations in the amount of antitoxin. It is interesting to note that this horse contained in its blood before receiving any diphtheria toxin an amount of antitoxin equal to two units per c.c. This is the largest quantity that we have met with. Whether this substance in normal blood should be considered a general or specific antitoxic substance is an interesting question which is now being investigated.

Date of Bleeding	Amount of Serum	Amount of Globulin	Amount of Diph. Antitoxin in each c.c.	Amount of Albumin
7–28–1899	10 c.c.	.3235	2 units in each c.c.	.3127
10–20–1899	10 c.c.	.8987	1200 units " " "	.2341
11– 9–1899	10 c.c.	.5934	650 units " " "	.2727
11–14–1899	10 c.c.	.5116	600 units " " "	.2938
11–18–1899	10 c.c.	.4292	400 units " " "	.3133

Between the first and second bleedings, weekly injections of strong diphtheria toxin were given; the last injection being 300 c.c. of a toxin of which 1/300 c.c. was the fatal dose of a guinea-pig of 250 grms. weight. Between the second and third bleedings, two injections of toxins similar in amount to the last noted were given. It is interesting to observe that, in spite of the continued toxin injections, the amounts of antitoxin and globulin both rapidly diminished. Between the last three bleedings no toxin was injected.

The above table gives a striking example of what Mr. Atkinson has found in all our horses. In no case has increase in globulin failed to accompany an increase in antitoxin.

These facts—the constant association of globulin with antitoxin, and the constant fairly proportional increase or decrease of the globulin in horses' blood with the increase or decrease of the antitoxin—bring the relation of these two substances so close together that while we are not yet fully justified in saying that antitoxic substances are globulins, still I think we must for the present at least consider that when we have obtained the globulin of the antitoxic blood we have obtained as nearly pure antitoxin as we have at present a prospect of getting.

I therefore decided to use this "antitoxic globulin" in cases of diphtheria, in place of the blood serum entire. The globulin with its antitoxin was obtained as follows: It was precipitated from the antitoxic serum by means of saturating it with magnesium sulphate, and then filtered off. The globulin was then pressed between a number of layers of filter paper, so as to remove as much of the saturated magnesium sulphate solution clinging to it as possible. The globulin and remaining magnesium sulphate were then dissolved in a quantity of water equal to one half the original bulk of serum from which the globulin

was obtained. The magnesium was then precipitated out as the phosphate (sodium magnesium phosphate) in the following manner: A little sodium chloride is first added, to prevent the precipitation of the magnesium as a hydrate, when the sodium hydroxide is added. Sufficient sodium hydroxide is then added to make the solution slightly alkaline. Finally the calculated amount of disodic hydrogen phosphate is added. The mixture is allowed to stand in a cool place, and stirred occasionally. Afterwards the precipitate of the magnesium sodium phosphate is filtered off. The filtrate contains all of the globulin and all of the antitoxin, together with about ten per cent. of sodium sulphate and a trace of sodium chloride. Each c.c. of the fluid will contain twice as many units as the original serum.

In practice I have diluted this antitoxic globulin so that each c.c. contained the same number of units as the original serum, as the concentrated solution seemed slightly more locally irritating.

Up to the present time, the globulin has been used in forty-eight cases, with the following results:

Locally, in about one fourth of the cases it causes some local œdema with slight pain — both somewhat more than the serum ordinarily produces.

Upon the diphtheria the results are equally good with those from the antitoxic serum. As to general effect, such as the rashes, results have been similar to those following the use of the serum.

Permanence.—The dried globulin has retained for two months the greater part of its antitoxic properties.

Solubility.—It is easily soluble, and can be readily put up in tablets, and so transported without danger.

Summary.—As a whole these results are very disappointing, as this antitoxic globulin apparently contains the greater part at least of those substances which cause the more or less deleterious effects of the blood serum, and as there seems no probability of separating the antitoxic properties from the globulin, the present outlook for a substance which, while being a specific neutralizing substance for diphtheria, will at the same time be absolutely harmless is not encouraging. What is true for diphtheria antitoxin is also probably true for other protective serums.

There seems to be some practical advantage, however, in having a soluble, permanent, and compact dry antitoxic substance which is not in danger of quick deterioration.

Further chemical experimentation will probably be able to show a practical method for separating the sodium sulphate from the globulin, if that seems desirable. Except for its slight local irritant effects, the sodium sulphate seems harmless.

ZUR AETIOLOGIE UND PATHOGENESE DER RACHITIS.

Von Professor A. MONTI,

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EINE grosse Anzahl hervorragender und erfahrener Autoren haben *unpassende Nahrung* im Verein mit den durch dieselbe bedingten Ernährungsstörungen als eine der wichtigsten Ursachen der Rachitis angesehen. Comby ist der Ansicht, dass die Rachitis meist der schlechten Ernährung im ersten Lebensjahre ihre Entstehung verdanke. Hoffmann nimmt an, dass zwar nicht jede beliebige Dyspepsie zur Entwicklung von Rachitis führt, dass aber ungenügende Nahrungszufuhr einerseits, mangelhafte Darmthätigkeit andererseits Rachitis veranlassen kann. Meinert anerkennt ebenfalls in den Ernährungsstörungen die wichtigste Ursache der Rachitis, nachdem bei guter und regelrecht gereichter Muttermilch die Erkrankung nie eintritt. Lamba, v. Stark, Fede, Mensi, Delmis, Zuccarelli nehmen ebenfalls einen alimentären Ursprung der Rachitis an. Besonders sucht Fede in unpassender Ernährung, gepaart mit schlechter Assimilation die Ursache zu Autointoxication und in der Folge zu Rachitis.

Auf Grundlage meiner Erfahrung muss ich ebenfalls die fehlerhafte Ernährung als Hauptursache der Rachitis ansehen, wenn dieselbe zu eigenthümlichen Störungen und Hemmungen der Spaltungs- und Oxydationsvorgänge führt, die wir noch näher beschreiben werden.

Alle unpassenden Nahrungsmittel, die den Organismus bis zu einem gewissen Grad ernähren, aber zu einer eigenthümlichen Form von Dyspepsie führen, bei der eine verminderte Bildung von Salzsäure und eine die normale Menge übersteigende Bildung von Milchsäure im Magensaft stattfindet, sind bei längerer Dauer geeignet, Rachitis zu verursachen.

Im ersten Lebensjahre können alle möglichen Nahrungsmittel diese Wirkung haben, während man früher geglaubt hat, dass dies nur der Fall sei, wenn die Nahrungsmittel specifische Veränderungen darbieten.

Zuerst hat man die Frage erörtert, ob in der *Frauenmilch* derartige specifische Veränderungen vorkommen, die geeignet wären, durch eine specifische Ernährungsstörung den rachitischen Prozess hervorzurufen.

Die Beantwortung dieser Frage ist äusserst schwierig. Hierzu ist zunächst die durch Wochen und Monate fortgesetzte tägliche Untersuchung der Milch einer Mutter nothwendig, da sich bezüglich ihrer Beschaffenheit zahlreiche Schwankungen ergeben, die nur durch fortgesetzte Untersuchungen festgestellt werden können, während eine ein- oder zweimalige Untersuchung nur einen ganz zufälligen Befund ergeben kann. Derartige Untersuchungen müssen überdies in den ersten Monaten der Stillperiode begonnen und womöglich bis zur Entwöhnung der Kinder fortgesetzt werden. Dabei sind zur Controlle die Ergebnisse der Körpergewichtszunahme und der übrigen Wachsthumfactoren, der Zustand der Verdauung und die sonstigen klinischen Erscheinungen des gestillten Kindes genau zu beobachten. Beginnt man mit der Untersuchung der Milch erst, wenn das Kind z. B. schon 5 Monate alt ist und bereits Erscheinungen von Rachitis zeigt, so ist kein beweisendes Resultat mehr zu erwarten, denn die Veränderungen der Milch, welche eventuell mit der Rachitis im Zusammenhange stehen, sind gewiss schon viel früher vorhanden gewesen und können in so später Zeit vielleicht schon normalen Verhältnissen Platz gemacht haben, so dass aus letzterem Umstande ein Rückschluss, dass die Milch auch früher normal war, ganz unstatthaft ist.

Solche mühsame Untersuchungen lassen sich allerdings an einem ambulanten Material nicht vornehmen; auch kann man nicht Mutter und Kind so lange in einem Spitale behalten. Man ist daher mit derartigen Untersuchungen auf die Privatpraxis angewiesen. Selbst da wird man auf viele Schwierigkeiten stossen, so dass sich nur einzelne Fälle verwerthen lassen und man erst nach einer Reihe von Jahren über das nöthige Material verfügen wird, um eine derartige Frage mit Sicherheit entscheiden zu können.

Pfeiffer hat bei 8 rachitischen Säuglingen 17 Muttermilchanalysen gemacht, indem er die Milch bei 2 Müttern je dreimal, bei 5 je zweimal und bei einer einmal untersuchte. Die Milchmengen wurden zwar nicht festgestellt, doch schienen sie Pfeiffer ausreichend. Die Zusammensetzung dieser Milchproben liess Pfeiffer keinen constanten Unterschied von der Norm erkennen und der Gehalt an Phosphorsäure zeigte sich in einzelnen Fällen um mehr als die Hälfte vermindert. Pfeiffer kommt zum Schluss, dass man keine bestimmte Abweichung der Muttermilch von der Norm als Ursache der Rachitis annehmen könne. Dass so wenige und in einem so späten Stadium vorgenommene

Untersuchungen für die vorliegende Frage nicht entscheidend sind, scheint Pfeiffer selbst anzunehmen, indem er schliesslich bemerkt, dass unter Umständen in der Milch rachitischer Mütter der Mangel an Salzen, besonders an Kalk und Phosphorsäure, sich in so ungünstiger Weise combiniren kann, dass die vererbte Constituitonsanomalie unter diesen Umständen beim Säuglinge leichter und intensiver zur Erscheinung kommen wird, als wenn die Nahrung von normaler Beschaffenheit wäre und geht so weit, auf Grund dieser Anschauung zu rathen, dass sobald die Analyse einen abnorm niedrigen Salzgehalt oder einen geringen Gehalt an Kalk und Phosphorsäure in der Milch der Mutter ergiebt, derselben das Selbststillen verboten werde.

Zander fand ebenfalls in der Milch von Müttern rachitischer Kinder ein Ueberwiegen der Kalisalze und Phosphorsäure über die Natronsalze und das Chlor und hält dieses Missverhältniss für die Ursache der Rachitis, indem in Folge dessen die Natronsalze und das Chlor unbenützt aus dem Organismus ausgeschieden würden. In Folge dessen soll es nicht zur Bildung einer hinreichenden Menge freier Salzsäure im Magensaft kommen, die zur Verflüssigung der durch das Labferment zur Verdauung vorbereiteten Eiweissstoffe und zur Lösung und Transportfähigkeit der Kalksalze nothwendig wäre. Dadurch tritt eine Aenderung der Resorptionsverhältnisse und eine verminderte Zufuhr derselben zu den Knochen ein und es kommt zu jenen Erscheinungen, die wir als Rachitis auffassen. Die verminderte Salzsäurebildung im Magensaft hätte auch die bei rachitischen Kindern fast constanten Störungen der Verdauung, Dyspepsien und Darmkatarrh zur Folge.

Seemann ist ebenfalls der Ansicht, dass die Verarmung der rachitischen Knochen an Kalksalzen nur die Folge einer specifischen Verdauungsstörung sei, bestehend in mangelhafter Salzsäurebildung im Magen. Diese sei wieder eine Folge von Mangel an Chloriden im Magen, der selbst wieder durch eine zu reichliche Zufuhr von Kalisalzen bedingt sei. Diese Hypothese stützt sich aber nur auf eine ganz unzureichende Zahl von Untersuchungen.

Nach meiner Ansicht handelt es sich bei der Frauenmilch nicht um specifische, sondern ganz gewöhnliche Veränderungen, die bei längerer Einwirkung keine acute Erkrankungen des Verdauungstractus hervorrufen, sondern eigenthümliche chronische Verdauungsstörungen bedingen, die zu einer verminderten Bildung von Salzsäure und zu vermehrter Bildung von Milchsäure im Magen Anlass geben. Allerdings verfüge ich über keine eigenen Untersuchungen über den Kalkgehalt der Frauenmilch und über das Verhältniss der Phosphorsäure, des Kali und Natron in derselben und kann daher die Anschauungen von Pfeiffer und Zander *a priori* nicht in Abrede stellen. Auf Grundlage

meiner vieljährigen Erfahrung scheinen mir jedoch gewisse Veränderungen der Frauenmilch während der Stillperiode bei längerer Einwirkung in bestimmter Form charakteristische Dyspepsien hervorzurufen, die auf die Knochenernährung eine bestimmte schädliche Wirkung ausüben.

Die natürliche Ernährung kann schon vom Momente der Geburt an eine fehlerhafte sein oder in jedem Alter des Säuglings eine solche werden, und die charakteristischen Verdauungsstörungen verursachen, sei es, dass dem Kinde gleich von der Geburt an eine schlecht beschaffene Frauenmilch dargereicht wird, sei es, dass die Milch dem Alter des Kindes nicht angepasst ist, indem es eine zu alte oder eine zu junge Milch bekommt, oder die Milch in irgend einem Stadium der Stillperiode wegen Erkrankung der stillenden Mutter oder Amme eine schlechte Beschaffenheit angenommen hat, sei es, dass eine an und für sich ganz entsprechende Frauenmilch durch zu langes oder zu oftmaliges Trinkenlassen im Uebermass gegeben wird, also Ueberfütterung stattfindet. Ja selbst über das eigentliche Säuglingsalter hinaus kann die natürliche Ernährung noch die Ursache dieser charakteristischen Störungen sein, wenn nämlich ein Kind zu lange, über den neunten Monat hinaus, gestillt wird, indem da die Frauenmilch schon zu wässerig ist, zu wenig Eiweiss und zu viel Zucker enthält, somit allein zur Ernährung des Kindes nicht mehr hinreicht und wegen des hohen Zuckergehaltes die bezeichneten krankhaften Erscheinungen hervorrufen kann. Nach meiner Erfahrung sind folgende Hauptmomente zu berücksichtigen.

Die Milch kann zu wässerig sein, zu wenig Fett enthalten und ein zu geringes specifisches Gewicht haben. Eine solche Milch dem Säugling lange gereicht bedingt eine zu geringe Körpergewichtszunahme, Anämie, Störungen der Magensecretion und wie bei allen Anämischen verminderte Salzsäureausscheidung. Da man in solchen Fällen oft durch zu häufiges Trinkenlassen, also durch Darreichung zu grosser Quantitäten, das Fehlende zu ersetzen sucht, so macht sich der Salzsäuremangel noch mehr geltend. Die Kinder vermögen die dargebotene Nahrung nicht normalmässig zu verdauen und in Folge Gährung des Zuckers bildet sich Milchsäure. So entsteht jene chronische Verdauungsstörung, die wochen- und monatelang anhält und zu verminderter Aufnahme von Kalksalzen, dagegen Aufnahme geringer Mengen Milchsäure in's Blut führt, welche beiden Momente als Ursache der Rachitis zu betrachten sind. Beginnt diese fehlerhafte Ernährung schon gleich nach der Geburt des Kindes einzuwirken, so können sich schon in der 6. bis 8. Lebenswoche die ersten Zeichen von Kraniotabes zeigen.

Ein zu hoher Fettgehalt der Muttermilch — über 3 % — mit gleichzeitig hohem specifischem Gewicht verursacht ebenfalls häufig Dyspepsien. Mit solcher Milch ernährte Kinder nehmen wohl zu, sind häufig sogar sehr fett, bleiben aber anämisch und fangen trotz normalen oder übernormalen Körpergewichtes schon nach dem zweiten Lebensmonate an, Zeichen von Rachitis zu zeigen.

Eine seltenere Veränderung der Frauenmilch ist ein zu hoher Zuckergehalt derselben. Am häufigsten kommt dies am Schluss der Stillperiode vor. Eine solche Milch einem Neugeborenen gereicht vermag er nicht zu bewältigen, sie vergährt und es bildet sich Milchsäure. Es entstehen Dyspepsien, Kolikanfälle, ungeheurer Meteorismus. Bald nimmt der Gehalt an Salzsäure ab, an Milchsäure zu, die Kinder werden anämisch, bleiben im Wachsthum zurück. Endlich stellen sich an Kopf und Brust die Erscheinungen von Rachitis ein.

Die gleiche Wirkung sah ich oft von Frauenmilch mit hohem specifischen Gewicht, reichlichem Eiweiss und ungenügendem Fettgehalt. Dies ist der Fall, wenn man zu einem Neugeborenen eine Amme mit sogenannter junger, d. i. 4—5 Monate alter Milch gibt. Eine solche Milch enthält für den Neugeborenen zu viel Casein und zu wenig Fett. Es bildet sich übermässig Milchsäure, es kommt zu Dyspepsien, endlich zu Erscheinungen von Rachitis.

Auch durch *frühzeitiges Entwöhnen* und Darreichung einer den Verdauungsverhältnissen des Kindes nicht angemessenen Nahrung können ähnliche zu Rachitis führende Störungen entstehen.

Die *künstliche Ernährung* der Säuglinge hat wohl in der Mehrzahl der Fälle Rachitis im Gefolge. Je mehr eine Methode der künstlichen Ernährung sich von der natürlichen entfernt, je unpassender also die Ernährung ist, um so sicherer verursacht sie Rachitis. Es würde zu weit führen, hier auf die einzelnen Methoden der künstlichen Ernährung näher einzugehen. Es sei nur bemerkt, dass im Säuglingsalter die ausschliessliche Ernährung mit Kaffee, Cacao, Amylaceis, Kindermehlen etc., ferner mit schlechter frischer oder condensirter Milch Störungen der Verdauung und Rachitis verursacht. Auch der sterilisirten Milch haften zahlreiche Mängel an. Bei Erhitzung auf 100° wird dem Kinde das Lactalbumin im geronnenen Zustande dargeboten, nachdem dasselbe schon bei 68°—74° gerinnt; ferner erfordert die sterilisirte Milch mehr Labferment um zu gerinnen und ist das Gerinnsel viel derber als das der einfach gekochten Kuhmilch. Die Verdauung des geronnenen Labfermentes und des derben Caseingerinnsels erfordert viel mehr Salzsäure als der Säuglingsmagen aufbringt. Ferner wird durch die Sterilisirung ein Theil der Kalksalze unlöslich gemacht und in Form eines Ueberzuges auf den Boden und die Wände der Flasche

niedergeschlagen. Nach Marfan reisst dieser Niederschlag auch die Hälfte der Phosphorsäure mit sich, und es ist noch nicht sicher erwiesen, dass ein Theil der ausgefallenen Kalksalze beim Abkühlen wieder in Lösung übergeht. Ferner kommt in Betracht, dass durch die Sterilisirung der Milch der Emulsionszustand der Fette zerstört wird und die Fettsäuren schwerer verdaulich werden. Trotz allem Enthusiasmus, der bei der Einführung der Sterilisation der Milch herrschte, wird die nüchterne Auffassung Platz greifen müssen, dass die Ernährung der Säuglinge mit Milch, die nach Soxhlets' Verfahren sterilisirt ist, vielfach Rachitis im Gefolge hat. Aber auch andere sogenannte physiologische Methoden der künstlichen Ernährung, wie nach Heubner-Hoffmann, nach Soxhlet, mit stark gezuckerter oder Fettmilch nach Gärtner oder Biedert haben nach meiner Erfahrung bezüglich der Entstehung der Rachitis die gleichen Nachtheile.

Alle angegebenen Methoden der künstlichen Ernährung veranlassen, je nachdem dabei mehr oder weniger Fehler begangen werden oder solche in sich schliessen, leichte oder schwere Rachitis. Nur die Ernährung mit Säuglingsmilch nach meiner Methode, wenn regelrecht durchgeführt, gewährt eine vollkommen tadellose Entwicklung der Kinder. Aber auch bei dieser Methode der künstlichen Ernährung kann ähnlich wie bei Brustkindern durch Ueberfütterung eine ungünstige Wirkung bis zur Entwicklung von Rachitis eintreten.

Verschiedene Autoren halten für eine weitere Ursache der Rachitis das plötzliche Aufhören der Ernährung mit Milch und den Ersatz derselben durch eine vorwiegend feste Nahrung aus Amylaceis und Fleisch. In dieser Richtung liegen interessante Versuche von Magendie und Guérin an jungen Hunden und von Pommay an jungen Vögeln vor.

Die Verdauungskraft des kindlichen Magens erreicht erst mit dem zweiten Lebensjahr jene des Erwachsenen. Es ist klar, dass ein frisch entwöhntes Kind vorwiegend feste Nahrung zu verdauen nicht im Stande ist und dadurch jene Störungen herbeigeführt werden, welche wir für die Entwicklung der Rachitis als geeignet bezeichnet haben. Ein Säugling, der plötzlich bei ausschliesslich fester Fleischnahrung oder Amylaceis entwöhnt wird, erkrankt im 9. bis 16. Lebensmonate unfehlbar an Rachitis.

Ueber die Pathogenese der Rachitis liegen zahlreiche Untersuchungen vor, die jedoch nach keiner Richtung genügen, um die Entstehung der Krankheit zu erklären.

Ueberblicken wir die in der Literatur vorliegenden Untersuchungen und Anschauungen über die Entstehung der Rachitis, so ergibt sich, dass uns Thierexperimente wohl den Weg gezeigt haben, auf welchem die Rachitis entstehen kann. Allein bei der Fütterung junger Thiere

mit Phosphor, Milchsäure, etc., mit oder ohne gleichzeitige Kalkentziehung werden künstliche Verhältnisse geschaffen, die von den bei Kindern obwaltenden wesentlich verschieden sind. So viel aber haben diese Experimente erwiesen, dass eine gewisse ins Blut gelangte Menge Milchsäure im Stande ist, einen schädlichen Einfluss auf die im regen Wachsthum begriffenen Knochen auszuüben, dass aber die dadurch bedingten Knochenveränderungen denen der Rachitis nicht gleichwerthig sind. Dazu muss noch ein zweites schädliches Moment, das der gestörten Kalkresorption einwirken.

Diese beiden Momente können bei unzweckmässiger Ernährung eintreten und so ohne verminderter Kalkzufuhr mit der Nahrung und ohne Einwirkung weiterer Schädlichkeiten Veränderungen hervorrufen, die wir als Rachitis bezeichnen.

Ich stelle mir den Vorgang der Entwicklung der Rachitis auf Grundlage der vorliegenden Forschungen und Experimente sowie der klinischen Beobachtung folgendermassen vor:

Unter dem Einfluss einer schon vorhandenen oder durch unpassende Nahrung hervorgerufenen Verdauungsstörung bildet sich im Magen eine etwas grössere Menge Milchsäure unter gleichzeitiger Verminderung der Salzsäure. Die zur Resorption gelangte Milchsäure übt einerseits einen Reiz auf die Knochengewebe aus, während anderseits in folge Verminderung der Salzsäure eine verminderte Resorption von Kalksalzen und ungenügende Zufuhr derselben zu den Knochen stattfindet. Dies wird noch im erhöhten Masse der Fall sein, wenn bedeutende Diarrhoe vorliegt, indem da der grösste Theil der Kalksalze unbenützt durch den Darm abgeht.

Der Einwand, dass grössere Mengen von Milchsäure im Magen fulminante Darmkatarrhe verursachen und so kaum Milchsäure in nennenswerther Quantität zur Resorption kommt, ist gegenstandslos. Solche Katarrhe bedingen allgemeine Abmagerung, endlich Atrophie der Kinder. Bei Rachitis handelt es sich nur um kleine Quantitäten Milchsäure, welche wohl zeitweise flüssige Stühle bedingen ohne die Resorption eines Theiles derselben auszuschliessen, umsomehr als wegen des verminderten Salzsäuregehaltes die Neutralisirung der Milchsäure durch die Kalksalze ausfällt und dadurch deren Resorption begünstigt wird. Damit es zu Rachitis kommt, darf diese Resorption von Milchsäure und verminderte Aufnahme von Kalksalzen ins Blut keine vorübergehende sein, sondern muss durch längere Zeit anhalten.

Die Beobachtungen von Vogel, Henoch u. A., die bei rachitischen Kindern zur Zeit der Untersuchung die Verdauung normal fanden, stehen mit meiner Anschauung nicht im Widerspruch. Wer die Entstehung der Rachitis bei einem Kinde genau zu beobachten Gelegenheit

gehabt hat, wird zugeben, dass jedenfalls zeitweise Meteorismus, Unregelmässigkeiten in der Defäcation, einmal Verstopfung, einmal flüssige Stühle und mangelhafter Appetit vorhanden sind. Dass Kinder mit zum Stillstand gekommener oder abgelaufener Rachitis normale Verdauung zeigen ist richtig, weil eben durch das Aufhören der Verdauungsstörungen die Bedingungen zum Stillstand oder zur Heilung der Rachitis gesetzt wurden.

Die Beobachtung Oppenheimer's, dass viele heftige Magen-Darm-Katarrhe ablaufen, ohne Rachitis im Gefolge zu haben, ist auf unsere Theorie nicht anwendbar, weil die Ursachen dieser Katarrhe wesentlich andere sind (Gährungs-Fäulnissprocesse) die eine ganz anders gestaltete Infection des Darmtractus bedingen.

Allerdings muss die von mir aufgestellte Hypothese erst durch künftige ausgedehnte Untersuchungen des Magensaftes, der Stühle, des Urins, selbst des Alkalescenz des Blutes begründet werden. Diese Untersuchungen müssen begonnen werden bevor die Rachitis manifest geworden ist und an Kindern mit florider, im Fortschreiten begriffener Rachitis fortgesezt werden. Untersuchungen an Kindern mit zum Stillstand gekommener oder abgelaufener Rachitis sind werthlos.

SECHS FÄLLE VON MALIGNEN NIEREN-TUMOREN IM KINDESALTER.

Von Dr. H. Rehn, Frankfurt a. m.

Wie mir der 8. internationale Congress in Kopenhagen durch die hochinteressanten Vorträge hervorragender Gelehrter (Pasteur, Virchow, u. A.), durch die glänzende Gastfreundschaft und die gebotenen Naturgenüsse, vor Allem der herrlichen Meerfarth nach Kronborg in frischester Erinnerung geblieben ist, so auch der animirte Verlauf der lehrreichen Verhandlungen in unserer Section für Kinderheilkunde, deren Leitung auf Wunsch und Vorschlag des liebenswürdigen Prof. Hirschsprung in die Hände unserer Freunde Jacobi und Rauchfuss gelegt war. Aber nicht allein in der musterhaften Leitung der Verhandlungen glänzten die beiden Ehren-Präsidenten, sondern noch mehr durch eigene, wissenschaftliche Verkündungen, so Rauchfuss durch seine musterhaften Vorträge über die "Berechtigung des klinischen Croup-Begriffs" und den "Werth der pädiatrischen Poliklinik auf die Verbreitung hygienischer Kenntnisse im Volke," und Jacobi durch einen ausgezeichneten Vortrag über das "primäre Nieren-Sarcom beim Foetus und im ersten Kindesalter." Unter Bericht über 4 eigene, anatomisch sichergestellte Fälle (worunter einer die Nierengeschwulst eines bei der Geburt verstorbenen Kindes betraf) und unter kritischer Durchmusterung aller in der Litteratur bekannt gegebenen Fälle, konnte Redner constatiren, dass das primäre Nierensarcom bei Kindern durchaus keine Seltenheit sei—entgegen der bisherigen Annahme—ja sogar beim Foetus angetroffen werde und in Häufigkeit seines Vorkommens das Carcinom bedeutend überrage.

An diese verdienstvolle Arbeit Jacobi's anknüpfend, welche wie so manche andere und Anderer in den Congressberichten ihr Grab gefunden hat,—denn ich finde sie weder in der Arbeit von Döderlein und Birch-Hirschfeld,[1] noch in der neuesten von Albarran[2] citirt—möchte

[1] "Embryonale Drüsengeschwulst der Nierengegend im Kindesalter." *Centralblatt für Krankheiten der Harn- und Sexual-Organe*, Bd. v., pg. 3 und 88–89, sq.

[2] "Néoplasmes du Rein," in *Traité des Maladies de l'Enfance*, publié sons la direction de Mon. Grancher, Comby et Marfan, vol. iii.

ich eine kurze Uebersicht über sechs, noch nicht publicirte Fälle von malignen Nierentumoren bei Kindern geben, trotz mancher Lückenhaftigkeit, und diesen kleinen Beitrag zur Festschrift dem Jubilar in aufrichtiger Hochachtung und herzlicher Freundschaft widmen.

Fünf der Fälle gehören eigner Beobachtung an; der sechste ist mir von meinem Bruder, Prof. Louis Rehn, zur Veröffentlichung überlassen worden.

Zur Charakterisirung dienen folgende Angaben:

1. Geschlecht. Befallen waren 4 Mädchen und 2 Knaben.

2. Lebensalter. Ein Kind war 1 J. 8 Mon., zwei 1 J. 9 Mon., eines 2 J., eines 5 und eines 11 Jahre alt.

3. In keinem Fall war ein hereditäres Moment nachweisbar.

4. In 5 Fällen wurde der Beginn der Erkrankung von den Angehörigen auf einige Monate und ein Jahr zurückverlegt. Bei dem 11jähr. Mädchen wollte man schon im 6. Lebensjahr einen aussergewöhnlichen Umfang des Leibes wahrgenommen haben.

5. Symptomatologie.

a. In allen Fällen handelte es sich um mässige Tumoren, welche sich von der Wirbelsäule zwischen unteren Rippen und Darmbeinkamm nach Innen und Unten bis zur Mittellinie, resp. darüber hinaus schoben. Die Oberfläche derselben war theils glatt, theils höckerig, einige Male waren grössere Knollen fühlbar, welche eine weiche Consistenz zeigten. Meist waren die Geschwülste von derber Härte.

b. Sie waren auffallenderweise sämmtlich *rechtsseitige.*

c. Haematurie war nur in 1 Fall (Prof. L. Rehn's Fall) beobachtet worden; in meinen 5 Fällen fehlte sie.

d. Die Harn-Menge war in keinem Fall auffällig vermindert. Eiweiss wurde nicht gefunden.

e. Als Drucksymptome kamen vor Oedem der rechten Unter-Extremität und Erweiterung der oberflächlichen Bauchvenen.

f. Metastasen, ausgedehnte, konnten in 2 Fällen während des Lebens in den Lungen nachgewiesen werden (Leere des Percussionsschalles, Fehlen des Athmungsgeräusches und des Stimmfremitus).

6. Zur Operation kam es in 2 Fällen (Mädchen von $1\frac{3}{4}$ und 5 Jahren) durch Prof. L. Rehn. Im 1. Fall nur zur Laparotomie. Nach Durchtrennung des Peritoneums präsentirt sich ein Tumor von theils blaurother, theils weisslicher Farbe, welcher den Respirationsbewegungen folgt. Die Oberfläche ist höckerig, von zahlreichen, dicken Venensträngen durchzogen; die bläulichen Stellen entsprechen Cysten. An dem medialen vorderen unteren Rand ist das Colon ascendens angeheftet.

Nach Oben und von der Mammillarlinie nach Aussen ist der Tumor

mit dem rechten Leberlappen flächenartig verwachsen; auch an der Porta hep. bestehen Adhäsionen. Am freien Rand desselben Lappens ein weisses, erbsengrosses Knötchen.

Natürlich wurde von der Exstirpation abgesehen. Zwei Tage später starb das ohnehin schon sehr herabgekommene Kind. Bei der Autopsie fanden sich zahllose Knoten an der Pleurae visc. und cost., vereinzelte erbsengrosse Knötchen in den Lungen; desgl. in der Leber. Rechte Niere in eine meist derbe, hier und da von Cysten durchsetzte Geschwulstmasse verwandelt; an einer Stelle der Peripherie noch normales Nierengewebe erkennbar. Linke Niere normal.

Ein Stück des Tumors wurde behufs mikroskopischer Untersuchung in das path. Institut (Prof. Weigert) abgesandt. (Ergebniss s. unten.)

In dem zweiten Fall, Mädchen von 5 Jahren, wurde von Prof. L. Rehn die Exstirpation des etwa Kindskopfgrossen Tumors vorgenommen, am 24. Sept. 1897. Der weitere Verlauf war ein sehr guter und wurde das Kind am 4. November in blühendem Aussehen und voller Munterkeit entlassen. Wundnarbe fest, nirgends Infiltration oder Schmerzhaftigkeit.

Allein schon am 4. Januar 1898, 2 Monate später, wurde das Kind in elendem Zustande wieder in das Krankenhaus gebracht. Es wurde ein Recidiv des Tumors an der früheren Stelle und Metastasen in der rechten Lunge constatirt, welche nur die Spitze freiliessen. Patient wurde alsbald in das Elternhaus zurückgebracht, wo es in Kurzem starb.

7. Path.-anatomische Diagnose der Tumoren.

Zu meinem grossen Bedauern liegt hier die schwache Seite dieser kleinen Veröffentlichung. In 1 Fall wurde die Section nicht gestattet.

Drei Fälle gehören der Zeit vor Weigert an. Hiervon sind einer als Carcinom, zwei als Sarcome bezeichnet; in einem der letzteren fand sich die anscheinend normale Niere plattgedrückt an der Unterseite der Neubildung, welche von dem perirenalen Bindegewebe oder von der Nebenniere ihren Ausgang genommen haben mochte. Der von Prof. L. Rehn exstirpirte Tumor ist gleichfalls als Sarcom bezeichnet.

Endlich verfüge ich aber doch noch über die eingehende histologische Untersuchung des Tumors, welcher der Leiche des laparotomirten Kindes entnommen wurde, aus dem path.-anatomischen Institut von Prof. Weigert. Ich verdanke dieselbe dem damaligen Assistenten, Dr. C. Herzheimer; sie lautet wie folgt:

(Färbung mit Haematoxylin und Picrocarmin.)

Dicke Bindegewebsstränge, mit theils mehr, theils weniger spindligen Zellen durchziehen das Präparat nach allen Seiten und geben

schmälere, untereinander communicirende Aeste ab, welche kleinere Hohlräume zwischen sich lassen, so dass das Ganze ein alveoläres Aussehen gewinnt. Innerhalb der Bindegewebsstränge sind vereinzelte typische Harncanälchen zu erkennen, ausserdem Gefässe und endlich einzeln oder zu Haufen atypisch liegende runde Zellen. Diese letzteren füllen auch, dichtgedrängt, die oben erwähnten Alveolen aus. Ihre Kerne erinnern an diejenigen der Epithelzellen der embryonalen Harncanälchen und sind der Mehrzahl nach rund, indessen finden sich auch ovale und etwas längliche darunter. Die Zellgrenzen sind nicht deutlich erkennbar. Von normalem Nierengewebe ist in den untersuchten Präparaten, die sämmtlich mitten aus den Tumormassen genommen waren, nichts zu erkennen.

Was hiernach die Diagnose anlangt, so kommen hier in Frage das Rundzellen-Sarcom und das Carcinom. Ersteres könnte man vielleicht vermuthen, da nicht sofort der epitheliale Charakter der obigen Geschwulstzellen in's Auge springt. Indessen sind dieselben vollkommen identisch mit den Epithelien der embryonalen Harncanälchen — und es ist dieser Fall, analog dem von Weigert (*Virchow's Archiv*, Bd. lxvii.) beschriebenen, als ein Carcinom zu betrachten, wofür überdies der exquisirt alveoläre Bau der Geschwulst spricht.

BEITRAG ZUR BEURTHEILUNG DES WERTHES DES BACCELLISCHEN VERFAHRENS BEI DER BEHANDLUNG DER ECHINOCOCCEN-CYSTEN. (ECHINOCOCCUS PLEURAE.)

Von Dr. JOHANN VON BÓKAY, Universitätsprofessor,

Director des Budapester Stefanie-Kinderspitales.

Es sind beiläufig drei Jahre verflossen, seitdem ich unter dem Titel: "Das Baccellische Verfahren bei Leberechinococcen der Kinder" eine kurze Mittheilung[1] bekanntgab, in welcher ich über den Verlauf dreier Fälle Bericht erstattete, die ich aus therapeutischem Zwecke mit *Baccelli*scher Punction behandelte. Das Verfahren, welches *Baccelli*, der hervorragende italienische Kliniker, zuerst im Jahre 1887 beschrieb[2] und bei dem in Rom im Jahre 1894 abgehaltenen internationalen Aerzte-Congress neuerdings anempfahl, hat sich bei meinen Fällen vollkommen bewährt, so dass ich auf Grund dreier Beobachtungen folgende Conclusionen ziehen konnte:

b. Das Baccellische Verfahren war in meinen drei Fällen von keinem schädlichen Einflusse auf den Organismus.

b. Die Echinococcuscyste begann in allen drei Fällen bald nach der Einspritzung zu schrumpfen, und einige Wochen nachher war die Anwesenheit der Blase nicht mehr constatirbar.

c. Ich sah keine Recidive der Cyste.

Es freut mich, dass ausser der europäischen Presse auch die amerikanische Fachpublicistik von dieser meiner kurzen Mittheilung Kenntniss nahm (*Jacobi*[3]), doch da auch seither meines Wissens keiner der Pädiatern diese Behandlungsweise versuchte, will ich jetzt mit einem erst vor Kurzem beobachteten Falle (diesmal ein Fall von Echinococcus pleurae) wieder vor die Öffentlichkeit treten, mit welchem ich den Werth des Baccellischen Verfahrens, und dessen grosse praktische Bedeutung neuerlich zu demonstrieren beabsichtige.

[1] *Archiv für Kinderheilkunde*, 1897.

[2] *Riform. med.*, 1887.

[3] *Therapeutics of Infancy and Childhood.* 2nd edition. Philadelphia, 1898.

Mein Fall ist der folgende:

K. T.—Knabe von 5 Jahren. Aufgenommen am 17ten Juli, 1899, in das Budapester "Stefanie"-Kinderspital.

Eltern und Geschwister sind angeblich gesund. Der Knabe war bis vor einem Jahre stets gesund als er an Pertussis, nachher an Pleuritis und vor einem halben Jahre an Pneumonie erkrankte. *Vor 4–5 Monaten wurde bei ihm in der linken Brusthälfte die Anwesenheit von Flüssigkeit constatiert*, deren Menge seit dieser Zeit ständig anwuchs. Anfangs hatte er Fieber, doch blieb dieses in letzter Zeit weg; er *hustete viel und zwar etwas erregt.* Der Appetit ist gut; auffallende Abmagerung. Vor einem Monate Morbilli; das Kind hat angeblich nie mit Hunden gespielt.—Das Kind wurde behufs eventueller Operation mit dem Verdachte von Empyema thoracis in das Spital geschickt. Bei der Aufnahme ist der Status präsens folgender:

Mässig entwickelter, *stark abgemagerter* Knabe, dessen Knochensystem die Zeichen einer abgelaufenen Rachitis aufweisen. Die Farbe der Haut ist normal, letztere wegen Schwinden des Fettpolsters überall leicht in Falten hebbar. Auf der Haut des Brustkorbes sind linksseitig mässige Venengeflechte sichtbar. Die Schleimhäute sind etwas blass. Der Kranke hält den Mund stets etwas geöffnet; die Sprache ist näselnd.

Der Brustkorb ist im Ganzen genügend gut gebaut, bei der Einathmung bleibt jedoch die linke Hälfte *merklich zurück; daselbst treten die Rippenzwischenräume mehr hervor*, währenddem sie rechts gut unterscheidbar sind; ebenso sind *die Gruben über und unter dem Schlüsselbeine links besser ausgefüllt.* Die Respiration ist sonst normal, von abdominalem Typus, kaum dyspnoëtisch, und erfolgt 44-mal in der Minute. *Der Herzstoss ist in dem*

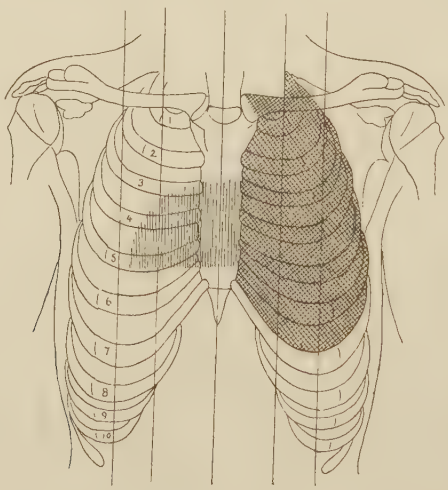

Fig. 1.

Scrobiculum sichtbar und fühlbar; die Herzthätigkeit ist ein wenig erregt; der Puls regelmässig, genügend voll; Zahl der Pulsschläge 112 in der Minute.

Bei der Percussion finden wir über der ganzen linken Brusthälfte überall eine *sehr intensive absolute Dämpfung*, welche sowohl vorne als rückwärts und auch an der Seite gleichmässig aufweisbar ist. Rechts ist der Percussionsschall sowohl vorne als hinten voll, hell und nicht tympanitisch, welcher als solcher in der rechten Parasternallinie bis zum oberen Rande der vierten Rippe, in der Mammillarlinie bis zum unteren Rande der sechsten Rippe, in der vorderen Axillarlinie bis zum unteren Rande der achten Rippe reicht; bei diesen

Grenzen beginnt die Herz-, respective Leberdämpfung. Rückwärts in der hinteren Axillarlinie finden wir den vollen, hellen, nicht tympanitischen Percussionston bis zum oberen Rande der zehnten, in der Scapularlinie bis zum oberen Rande der eilften Rippe. *Die Herzdämpfung ist stark nach rechts und unten verlagert.* Die Grenzen der absoluten Herzdämpfung sind oben der obere Rand der vierten Rippe, rechts der rechte Rand des Brustbeins, links confluirt dieselbe mit der über der Lunge gewonnenen Dämpfung; die relative Herzdämpfung reicht rechts beinahe bis zur rechten Mammillarlinie. (Siehe Figur I.)

Mittelst Auscultation bemerken wir, dass über der rechten Lunge überall das Athmen normal, etwas rauh klingt, während linkerseits sozusagen absolut kein Respirationsgeräusch hörbar ist. Herztöne und die der grossen Gefässe sind rein, genügend kräftig. Der Bauch ist nicht angetrieben. Die Milzdämpfung confluirt mit dem dumpfen Schalle der Brusthöhle; bei tiefer Inspiration ist die Milz in der Höhe des Rippenbogens fühlbar, doch nicht vergrössert. Die Lebergrenze überschreitet den Rippenbogen in der Mammillarlinie mit einer Fingerbreite. Die Function der Bauchorgane ist normal.

Bei der Aufnahme ist der Kranke fieberfrei; Allgemeinbefinden zufriedenstellend.

Am Tage der Aufnahme machte der Abtheilungsarzt bei der Abendvisite eine Probepunction zwischen der sechsten und siebenten Rippe in der linken hinteren Axillarlinie; das Resultat derselben war eine vollkommen wasserklare, durchsichtige Flüssigkeit. Damals fiel bereits auf die vollkommene Klarheit der Flüssigkeit, sowie jener Umstand, dass sich dieselbe unter grossem Drucke entleerte, so zwar, dass sich die Punctionsspritze von selbst anfüllte, ohne dass an derselben hätte gezogen werden müssen. Nichtsdestoweniger nahm der behandelnde Abtheilungsarzt die gewonnene Flüssigkeit für exsudirtes Serum (die geringe Menge der erlangten Flüssigkeit — 2–3 Cm. — eignete sich nicht für eine genauere Untersuchung), und innerlich wurde Nat. salicylicum verordnet.

Des anderen Tages trat geringes Fieber auf (38.6°).

Eine Woche nach der Aufnahme war das Ergebniss der physikalischen Untersuchung ein unverändertes, bei gutem Allgemeinbefinden war der Knabe fieberfrei, die Athmung in geringem Grade erschwert, das supponirte Pleuraexsudat unvermindert. Aus diesem Grunde beschloss ich die partielle Abzapfung, welche ich behufs der Verbesserung der Thoraxdruckverhältnisse am 26ten Juli mit *Potain*schem Apparate vollzog. Bei dieser Gelegenheit entleerte ich 370 Cubiccm. durchsichtige, farblose Flüssigkeit, welche durch winzig kleine, Luftbläschen ähnliche Körnchen etwas getrübt erschien. Die Flüssigkeit verwies schon bei makroskopischer Betrachtung auf die Anwesenheit einer Echinococcusblase; die genaue Untersuchung ergab unzweifelhaft den Beweis der Richtigkeit obiger Annahme. Das specifische Gewicht der Flüssigkeit war *1010* Reaction neutral; *Kochsalzgehalt bedeutend, dagegen nur verschwindende Spuren von Albumen;* Zucker ist in derselben mit Trommerscher Probe nicht nachweisbar. Nach zwölf Stunden bildete sich ein *beträchtlicher* Niederschlag; in demselben sind *mikroskopisch sowohl einzelne Echinococcus-Häkchen als auch selten schöne Scolexgruppen in grosser Anzahl auffindbar.*

Nach der Punction verminderte sich die Intensität der Dämpfung über der linken Thoraxhälfte, und blos über dem oberen Theile des Brustkorbes bis zum mittleren Drittel der interscapularen Region erhalten wir absolut klanglosen Percussionsschall. In der Gegend der Punction auf handtellergrossem Gebiete tritt unter mässigen Fieberbewegungen Schmerzhaftigkeit, Röthung und subcutanes Emphysem auf; der Kranke hustet viel, das *Sputum ist jedoch nicht blutig.* Das Fieber sowie das subcutane Emphysem schwinden binnen 3 Tagen, auch der Hustenreiz wird seltener.

Am 3ten August reicht die absolute Dämpfung vorne bis zum dritten Intercostalraum, rückwärts bis zur Spina Scapulæ; unter diesen Grenzen erhalten wir einen gedämpft tympanitischen Percussionsschall. Die Auscultation ergiebt rückwärts abgeschwächtes, vorne sowohl als hauptsächlich in der Achselhöhle bronchiales Athmen.

Am 4ten August operativer Eingriff nach *Baccelli*s Methode. In der linken Achselhöhle, zwischen der vierten und fünften Rippe Punction mit Lüers Glasspritze, und

Entleerung von *30 Cubiccm.* Flüssigkeit, die der bei der früheren Punction gewonnenen vollkommen ähnlich ist; dieselbe entleerte sich wieder unter so grossem Drucke, dass die Spritze sich von selbst anfüllte; hernach spritzte ich circa *20 Cubiccm.* filtrierte 1_{00}^{0} Sublimatlösung ein. Dieser Eingriff hatte fünf Tage hindurch Fieberbewegungen mit dem Maximum von 39.2 ° C. zur Folge; ausser dem Fieber war jedoch weder eine allgemeine, noch eine locale Reaction bemerkbar. Das Allgemeinbefinden des Patienten blieb dauernd ein zufriedenstellendes.

Am 11ten August war der Lungenbefund folgender: Der Percussionston ist über der ganzen linken Thoraxhälfte dumpf, und geht nur hinten von der Mitte der Scapula abwärts

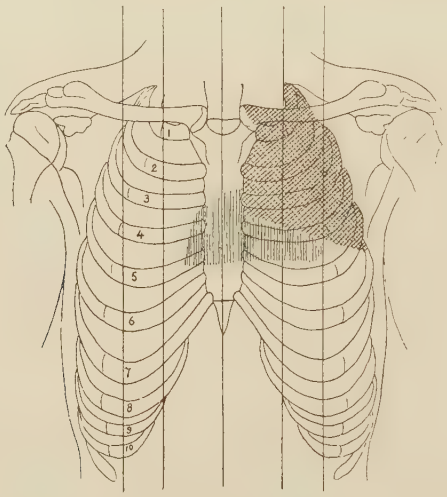

Fig. 2.

in gedämpft tympanitischen Ton über. Auscultation ergiebt über der ganzen linken Brusthälfte bronchiales Athmen, tief unter der Achselhöhle ist amphorisches Athmen hörbar. Über der rechten Brusthälfte normale Verhältnisse. Patient ist andauernd fieberfrei.

Am 30ten August, drei Wochen nach der *Baccelli*schen Punction, ist der Percussionsschall vorne bis zum dritten Intercostalraum, hinten im Interscapularraum bis zur Höhe des fünften Rückenwirbels intensiv gedämpft, von hier abwärts bis zum unteren Rande der achten Rippe wenig gedämpft, tiefer unten tympanitisch; bei der Auscultation ist vom unteren Drittel der Scapula abwärts schwaches amphorisches Athmen noch vernehmbar. Das Herz ist nunmehr blos wenig dislocirt; die Herzdämpfung fliesst theilweise mit der über der Lunge vorhandenen Dämpfung noch zusammen, überschreitet nach rechts den rechten Sternalrand um 1 Cm.; der Spitzenstoss ist in der linken Mammillarlinie. (Siehe Figur II.) Verbleibt ohne Medicament.

Am 2ten September zweite *Baccelli*-Operation. Die in der Höhe der fünften bis sechsten Rippe im Interscapularraume vorgenommene Probepunction hatte ein negatives Resultat; hierauf wurde in der mittleren Achsellinie im zweiten Intercostalraume die Punction vollführt und circa *30 Cubiccm.* blassgelbe getrübte dünnflüssige Flüssigkeit entleert und beinahe *20 Cubiccm.* 1_{00}^{0} Sublimatlösung injiciert. Der Kranke überstand auch diesen Eingriff vollkommen gut ohne Reaction.

Bei fortdauerndem fieberfreien Befinden besserten sich die physicalischen Zeichen der Brusthöhle allmählig. Am 11ten September war über der linken Lungenspitze sowohl vorne als hinten stark gedämpfter tympanitischer Percussionsschall zu finden, währenddem in der Achselhöhle ein ausgeprägt tympanitischer, nur kaum gedämpfter Percussionston zu finden

war. Die Dämpfung verkleinert sich fortwährend, am 30ten September ist bereits nicht nur in der Achselhöhle, sondern auch über der Lungenspitze vorne und hinten der etwas gedämpfte Schall stark tympanitisch gefärbt; auf diesem Gebiete ist abgeschwächtes, sonst überall normales rauhes Athmen zu vernehmen. Die Herzdämpfung ist nunmehr nicht dislocirt, reicht nach oben bis zum zweiten Intercostalraum, nach rechts bis zum rechten Rande des Brustbeines, nach links bis zum Orte des Spitzenstosses, welcher zwischen der vierten und fünften Rippe etwas ausser der Mammillarlinie auffindbar ist. Allgemeinbefinden und Appetit sind gut; Patient hustet kaum.

Im Monate Oktober weist der physicalische Untersuchungsbefund sowohl die Ausbreitung

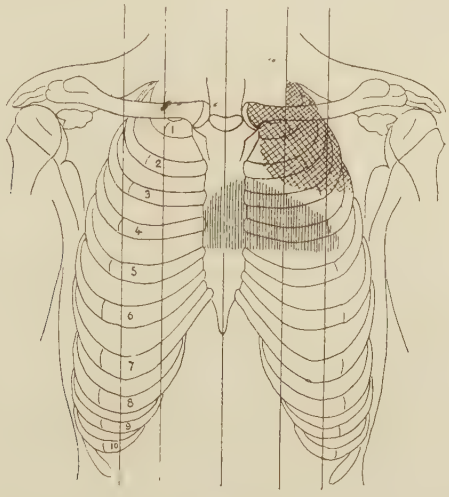

Fig. 3.

der Dämpfung, als auch deren Intensität betreffend, neuerdings eine wesentliche Besserung auf, so dass am Schlusse dieses Monats der Percussionsschall nunmehr etwas gedämpft ist, und hinten die Spina scapulæ abwärts mit einem Centimeter überschreitet. Über der ganzen Lunge war blos rauhes Athmen hörbar, mit einigen knarrenden Rasselgeräuschen. Herzgrenzen normal. (Siehe Figur III.) Fieberfrei.

Auch diese geringe percutorische Abweichung verschwand bald, so dass am 6ten November vorne überhaupt mehr keine Dämpfung constatirbar war, blos hinten über der Scapula der Percussionsschall noch etwas gedämpft ertönt.

Der physicalische Befund war der gleiche, als ich den Kranken am 26ten November, daher nach 4½-monatlicher Spitalsbehandlung, entliess; das Körpergewicht des Kranken hatte während dieser Zeit mit mehr denn *3 Kilogramm* zugenommen.

In meinem vorhin detaillirten Falle supponierte ich einen *primären Pleura-Echinococcus*, ich muthmasste daher eine solche Localisation der Echinococcusblase, welche laut unseren litterarischen Kenntnissen eine der grössten Seltenheit ist. (Neisser[1] hat unter *983* aus der Litteratur zusammengestellten Echinococcusfällen primären Pleura-Echinococcus blos in *17* Fällen, daher in *1.73 %* der Beobachtungen gefunden.) Es ist wahr, dass die Begleiterscheinungen des Echinococcus pleuræ, namentlich die von den Autoren im Allgemeinen betonten: Seiten-

[1] A. Neisser. *Die Echinococcen-Krankheit.* Berlin, 1877.

stechen, Dyspnoe, quälender Husten in meinem Falle nur kaum ausgeprägt war, die Qualität der Untersuchungsflüssigkeit, sowie die von der Percussion und Auscultation gelieferten Symptome waren jedoch so überzeugend, dass überhaupt kein Zweifel auftauchen konnte, dass wir mit einem intrathoracal gelegenen Echinococcus-Sacke zu thun hatten. Echinococcus der Lunge glaube ich wegen Mangel des quälenden Hustenreizes und Abwesenheit eines sanguinolenten Sputums ausschliessen zu können, obzwar ich dessen bewusst bin, dass es wohl zumeist unmöglich ist zwischen Echinococcus der Lunge und der Pleura symptomatisch eine scharfe Unterscheidung zu stellen.[1] Zur Annahme eines primären Pleura-Echinococcus berechtigte mich jener Umstand, dass ich die übrigen Organe, und insbesondere die Leber während der ganzen Beobachtungsdauer intact fand.

In unserem Falle kamen die Eier wahrscheinlich auf dem Wege der Lymphbahnen auf die Oberfläche der Pleura, und zwar intrapleural, zwischen die Blätter des visceralen und parietalen Brustfelles, so begann die Entwicklung der Blase, vermuthlich in der Richtung von oben nach unten. Jener Umstand, dass nach der ersten *Baccelli*schen Punction ein mässiger, den Kranken kaum belästigender Pneumothorax auftrat,[2] scheint zu beweisen, dass die Compression der Lunge der Längenachse entsprechend erfolgte, und dass die Nadel der Spritze bei dem Einstiche Alveolen verletzte.

Das *Baccelli*sche Verfahren hat sich auch in diesem unseren Falle bewährt, es enthellt nämlich unzweifelhaft aus dem Krankheitsverlauf, dass die ersten Symptome der Besserung bald nach der Sublimatinjection auftraten, und dass der rasche Fortschritt der Heilung besonders nach der zweiten Einspritzung ein auffälliger war. Mein Fall ist daher, obzwar ich ihn derzeit noch nicht als vollkommen geheilt betrachten kann, ein neuer Beweis des Erfolges der *Baccelli*schen Methode und *der erste Fall in der Litteratur, welcher bezeugt, dass dieses Verfahren nicht nur bei Echinococcus der Leber sondern auch bei intrathoracaler Lagerung der Echinococcusblase zum Erfolge führt.* Der praktische Werth dieses unbedeutenden Eingriffes gewinnt noch mehr, wenn wir vor Augen halten, dass wir durch dieselbe eine sehr eingreifende, ernste, blutige Operation überflüssig machten. Jener Umstand, dass Cornil, Gibier sowie Schede schon nach einfacher Punction[3] des intrathoracalen Echinococcus tödtlichen Ausgang auftreten sahen, kann den Werth des Baccellischen Verfahrens nicht verringern.

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[1] Riedinger. "Verletzungen u. Krankh. des Thorax." *Deutsche Chirurgie.*

[2] Ich bin geneigt das am 11ten und 30ten August notirte amphorische Athmen dem Auftreten eines geringgradigen Pneumothorax zuzuschreiben.

[3] Riedinger, *l. c.*

DIE NORMALEN GRENZEN DER GROSSEN UND KLEINEN HERZDÄMPFUNG IM KINDESALTER.

VON DR. MED. J. W. TROITZKY,

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DIE Frage von den normalen Grenzen des Herzens in verschiedenen Perioden des Kindesalters kann man noch längst nicht für endgültig entschieden erklären, ungeachtet einer ganzen Reihe in dieser Hinsicht gemachter Untersuchungen und streng wissenschaftlich erläuterter klinischer Beobachtungen. Die ungeheure mit Nichts vergleichbare Bedeutung des Herzens, als eines Organes, welches nicht nur tonangebend für alle Lebensprocesse, sondern auch bedingend für dieselben ist, muss eine besondere Aufmerksamkeit auf sich lenken zur Zeit des Wachstums des menschlichen Organismus, wo das geheimnissvolle Leben dieses edelsten Apparates noch complicirter wird, in Anbetracht der Notwendigkeit einer selbstständigen Entwickelung in Hinsicht auf Grösse und Gewicht. Es wird jedoch das grosse Interesse beim Studieren des kindlichen Herzens in hohem Grade paralysirt durch die Schwierigkeit der Untersuchungen und durch die Eigentümlichkeiten beim Wachsen, das nach seinen eigenen Gesetzen vor sich geht, unabhängig von den Regeln, nach welchen sich die Gewichtszuname des Körpers gestaltet und dessen Längenwachstum.[1] Diese Eigentümlichkeiten müssen eine hervorragende Rolle bei der Bestimmung der Grenzen der Herzdämpfung spielen, welche völlig von Mass und Grösse des untersuchten Organs abhängt und nur teilweise vom Alter des Kindes, von dessen Gewicht und der Länge des Körpers. Wenn der Umfang des Brustkastens und insbesondere seiner vorderen Oberfläche in Abhängigkeit steht von der Länge und dem Gewicht des Körpers, wobei in einem bestimmten Alter diese Oberfläche unter normalen Bedingungen bei der Entwickelung, eine bekannte, mehr oder weniger bestimmte Grösse haben muss, so folgt daraus noch bei Weitem nicht, dass auch die Herzgrenzen in gleichem Mass abhängig sind von diesen Verhältnissen. Die Brust kann dann an Umfang zunehmen, während

[1] Siehe den Aufsatz im *Archiv für Kinderheilkunde*, Bd. xxvi, 1–2: "Ueber die Bedeutung der Eigentümlichkeiten des wachsenden Organismus bei Dosirung der Arzneimittel."

das Herz fast unverändert bleibt, im Sinne von Umfang und Gewicht. Andererseits ist eine vermehrte Entwickelung des Herzens durchaus möglich bei schwachem Wachstum des Brustkastens, ja sogar bei zeitweiligem Stillstand in der Entwickelung. Im ersten Fall werden die Grenzen der Herzdämpfung verhältnissmässig kleiner sein, im zweiten — bedeutend grösser, wobei die bestimmten Linien und Puncte nicht dienen werden als beständige hinweisende Momente für den vom Herzen eingenommenen Raum in der Brust. Dass die Curven des Herzwachstums an Umfang und Gewicht scharf sich unterscheiden von dem mittlern Wachstum des Körpers, davon kann man sich leicht überzeugen auf Grundlage der Tab. *C* und *D* der eben citirten Arbeit und der Tab. *C* meiner vorhergehenden Untersuchungen.[1]

Es erweist sich z. B., dass das Körpergewicht sich bis Ende des 5. Monats verdoppelt, die Körperlänge im 7. Jahr, der Umfang des Herzens wird zweimal so gross am Ende der Säuglingsperiode, während das Gewicht des Herzens in derselben Zeit um 2½mal sich vergrössert. Die Verdreifachung des Körpergewichtes tritt ein am Ende des ersten Jahres, die der Körperlänge nach dem 17. Jahr, die des Herzumfanges im 4. Jahre, die des Herzgewichtes im 3. Jahr. Vor mehr als 60 Jahren hat Bizot[2] die Aerzte bekannt gemacht mit den Daten bei Messung des Herzumfanges in verschiedenen Perioden des kindlichen Alters. Aus diesen Daten ist ersichtlich, dass die Herzlänge in den ersten 4 Jahren des Lebens sich um 5.12 Ctm. vergrössert, im 5. bis 9. Jahr um 6.5 Ctm., und im 10. bis 15. Jahr um 7.18 Ctm., und die Breite, in derselben Reihenfolge, ist in folgender Weise ausgedrückt: 5.96, 6.99, 7.69 Ctm. Den grössten Umfang bei Kindern hat das Herz nach Beneke[3] während des ersten Lebensjahres und dann wird es immer kleiner im Verhältnisse zum Körpermass, bis zum Anfang der Geschlechtsreife, wobei in den ersten 5 Jahren der Umfang sich nicht ändert, während die Gewichtszunahme seinerseits vorwärts geht.[4] Der Umfang des Herzens wächst nicht proportional dem Alter, indem er fast sich gleich bleibt von der Hälfte des 2. Jahres bis zum 5½ Jahre und darauf unregelmässig sich vergrössernd bis zur Periode der Geschlechtsreife.[5]

Die hier angeführten Daten genügen wie mir scheint vollständig,

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[1] "Die Dosirung der Heilmittel im Kindesalter." *Kiewer Universitäts-Nachrichten*, 1893.

[2] H. Sahli, W. von Starck, E. Weill. S. u.

[3] *Die anatom. Grundlagen der Constitutionsanomalien des Menschen.* Marburg, 1878.

[4] C. Hochsinger. "Die Auscultation des kindl. Herzens." *Beiträge zur Kinderheilkunde*, von M. Kassowitz, II. Heft, 1890. Wien. § 7.

[5] E. Barthez et A. Lanné. *Traité clinique et pratique des maladies des enfants.* T. i, pag. 54. 1889. Paris.

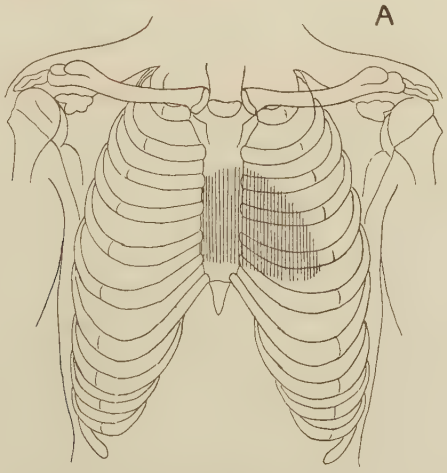
als Beweise für die oben ausgesprochenen Behauptungen, wie auch als Erklärung der von einander abweichenden Meinungen der Autoren in Bezug auf die normalen Herzgrenzen in verschiedenen Perioden des Kindesalters. Bei der weiter folgenden kurzen Auseinandersetzung in Bezug auf die Arbeiten über den vorliegenden Gegenstand werden wir deutlich namhaft machen einige auseinandergehende Meinungen der Autoren, ihre Ursachen, und den Wegan geben, an welchen sich haltend man, wenn auch noch nicht augenblicklich, so in nicht weiter Zukunft eine völlige Uebereinstimmung erreichen kann und weitere Streitpunkte aufheben kann in einer Frage, deren Entscheidung ein bedeutendes Interesse bietet.

Gierke[1]: Die Resultate von 50 Beobachtungen bei Kindern im Alter von 8 Tagen bis 13 Jahre. Das Herz liegt in toto bei Kindern höher, auf Grund des Höherstehens des Zwerchfells.

Die Herzspitze befand sich in 38 Fällen im 5. Zwischenrippenraum, in 6 Fällen im 6., 2mal im 4., 3mal auf der 4. Rippe und einmal auf der 6.

Die Grenze der Dämpfung links und unten: in 44 Fällen in der Brustwarzenlinie, in 4 Fällen ausserhalb und in 2 Fällen innen von dieser. Ausserhalb maximum 1 Ctm., innerhalb 1.5 Ctm. Die untere Horizontalgrenze überragt rechts ein wenig die mittlere Brustbeinlinie.

Die Länge der grossen Dämpfung, gerechnet von der Stelle des Herzstosses bis zur



Mitte der rechten Linie der Dämpfung: Säuglingsalter, 5—8.5 Ctm.; von 1—3 J., 6.5—9 Ctm.; von 3—4 J., 7.5—9.5 Ctm.; von 4—6 J., 9.5—11 Ctm.; 6—10 J., 9—12 Ctm.; 10—13 J., 9—12 Ctm. Die kleine Dämpfung im Alter von 3—8 J., 2.25—4 Ctm.; in der Horizontallinie, 2.5—4 Ctm. (Zeichnung *A*.)

A. Steffen[2]: Die Herzspitze liegt auf der Brustwarzenlinie, manchmal ausserhalb derselben, aber nicht mehr als 1 Ctm.

[1] "Ueber die Lage und Grösse des Herzens," *Jahrbuch für Kinderheilkunde*, 1889, Bd. ii., S. 4.

[2] *Ibidem*, Bd. iii., 1870, "Beitrag zu der Lehre von den Herzkrankheiten."

Die obere Grenze der grossen Herzdämpfung ist die linea mediaana sterni, seltener um 0.5—1.5 Ctm. links davon und noch seltener um 0.5—1 Ctm. rechts. In Bezug auf die Rippen der 2. Intercostalraum, in anderen Fällen der obere Rand der 2. und sogar 3. Rippe.

Rechts geht die Grenze fast vertical nach unten und ist von der mittleren Linie um 1—3.75 Ctm. entfernt, in den Grenzen des unteren Randes der 4. Rippe oder des oberen Randes der 5. Rippe.

Das Verhältniss der Breite der grossen Herzdämpfung zur Länge derselben kann ausgedrückt werden durch 2:3. Die kleine Dämpfung hat bald die Form eines Dreieckes mit der Grundlinie unten, bald die eines Viereckes, dessen Seitenlinien länger als dessen Horizontallinien sind. Das Dreieck pflegt selten gleichmässig zu sein, gewöhnlich ist die Höhe desselben bedeutender als die Grundlinien. (Zeichnung *A*.)

A. Stössl[1]: Das Herz liegt bei Kindern anfangs horizontal und nimmt während des Wachsens die Lage ein, die es bei Erwachsenen hat.

Die Spitze befindet sich auf der Warzenlinie, selten 0.5—1 Ctm. aussen von dieser. Je grösser das Längenmass des Brustkastens, desto tiefer steht die Spitze, je bedeutender das Quermass, desto höher. Bei normalen Verhältnissen im 5. Rippenzwischenraum, bei breiter Brust zwischen 4. und 5. Rippe, bei langer im 6. Intercostalraum.

Die obere Grenze der grossen Dämpfung links der 2. Zwischenrippenraum oder der obere Rand der 2., seltener 3. Rippe, oben die linea mediana sterni oder 0.5—1 Ctm. rechts oder links davon. Rechts geht die Dämpfungslinie schräg über den 4. Intercostalraum, den untern Rand der 4., selten der 5. Rippe.

Th. von Dusch[2]: Das Herz liegt mehr quer, entsprechend dem höhern Stande des Zwerchfells.

Der Herzstoss befindet sich im 5. Intercostalraum, in der linea mamillaris oder links von derselben, selten auch rechts. Bei Kindern über 3 J. ist der Stoss deutlich bemerkbar, in der allerersten Kindheit ist er nicht deutlich und sogar schwer durchzufühlen, wegen der geringen Breite der Intercostalräume.

Die obere Grenze der grossen Dämpfung links ist der 2. Intercostalraum oder das Brustende des 3. Rippenknorpels. Die linke Grenze geht kreisförmig nach aussen, schneidet im 5. Zwischenraum die Warzenlinie und steigt ab links von der Stelle des Herzstosses. Rechts geht die Dämpfung ein wenig über den Rand des Brustbeines hinüber in der Höhe der 3., 4. und 5. Intercostalräume. Die obere rechte Grenze geht nach unten in absteigender Richtung.

K. A. Rauchfuss[3]: Uebereinstimmend mit den Daten von Scoda und Luschka stehen sowohl das Zwerchfell, wie auch das Herz höher als bei Erwachsenen. Die Lage der Längsaxe des Herzens ist eine mehr horizontale. Bei Neugeborenen sind der hintere, quere und vordere Durchmesser des Brustkastens einander gleich, im 6. J. steht der erste zum zweiten im Verhältniss von 1:0,8, im 10—12 J. wie 1:0,7, bei Erwachsenen wie 1:0,7. Diese Eigenthümlichkeit kann als Grund dienen für die Abweichung der Herzspitze nach aussen. Bei der Bestimmung der Grenzen der Dämpfung sind das Herz, die Warze und die Warzenlinie weniger massgebend; auf Grund ihrer Veränderlichkeit ist es wünschenswert, sich bei derartigen Untersuchungen an die Mittellinie, die Rippen und Intercostalräume zu halten.

Die Herzspitze liegt höher, als bei Erwachsenen, um einen ganzen Intercostalraum oder etwas weniger. Diese Eigentümlichkeit betrifft hauptsächlich die ersten drei Jahre des Lebens, wobei man immer die individuellen Schwankungen im Auge behalten muss. In

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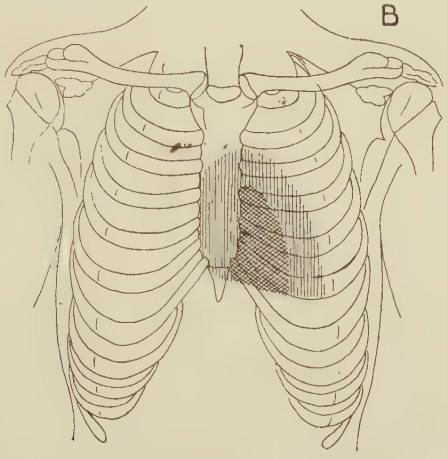
[1] *Semiatik und Untersuchung des Kindes*, Stuttgart, 1875, S. 153—155.

[2] "Die Krankheiten des Myocardium." *Gerhardt's Handbuch der Kinderkrankheiten*, Bd. iv., 1. Abth., S. 276—279, Tübingen, 1878.

[3] "Zur physiologischen Untersuchung des Herzens." *Gerhardt's Handbuch*, iv. Bd., 1. Abth., 1878, S. 3—11.

Bezug auf den Herzstoss liegt die Spitze um 1 Ctm. nach aussen und unten, wobei der erste die Warzenlinie nach aussen um 0.5—1 Ctm. überragt, während die Spitze dieselbe um 1—2 Ctm. überragt. Es ist sogar ein Ueberragen nach aussen von der linea mamillaris um 2 Ctm. möglich, in Abhängigkeit von den Eigentümlichkeiten des Wachstums. Diese Erscheinung kommt oft vor bei Kindern von 10—12 Jahren bei normaler Entwickelung.

Den Herzstoss kann man mit dem Auge wahrnehmen im 4. und 5. Intercostalraum oder in beiden zusammen, zwischen der linea mamillaris und parasternalis, aber näher zur ersten, ja



auf ihr selbst oder mehr links. In den ersten Lebensjahren kann der Stoss ganz fehlen, wegen der geringen Breite der Intercostalräume.

Die grosse Dämpfung beginnt oben und links vom Knorpel der 2. Rippe oder des 2. Intercostalraumes, geht kreisförmig nach links und unten bis zur 6. Rippe, indem sie die 3. Rippe oder den Knorpel auf der linea parasternalis, die 4. Rippe auf der linea mamillaris, die 5. Rippe 0.5—1 Ctm., die 6. Rippe 1—2 Ctm. aussen von der Warzenlinie schneidet. Die rechte obere Grenze liegt in der Höhe der absoluten Leberdämpfung. Die untere Grenze ist horizontal und erstreckt sich von dem untern Rand der 6. Rippe oder des 6. Intercostalraums in der Nähe des rechten Randes des Brustbeins bis zum untern Rand derselben Rippe oder Intercostalraums der linken Seite um 1—2 Ctm. ausserhalb der linea mamillaris. Die rechte Grenze oben geht vom 2. Zwischenrippenraum oder vom 3. Rippenknorpel kreisförmig nach unten, nach aussen von dem Brustbein, wobei auf der Höhe der Zwerchfellkuppel sie am meisten entfernt liegt von dem Rande des sternum, an diesem Punkt beinahe die linea parasternalis erreichend.

Sich nach unten begebend, geht die rechte Grenze allmählig zum Brustbein. Die Länge der verhältnissmässigen Dämpfung erstreckt sich von der Herzspitze bis zum Brustrand des 2. rechten Rippenknorpels. Die kleine oder absolute Dämpfung geht von der Stelle der Anheftung der 4. linken Rippe an das Brustbein nach unten bis zur 7. zur linea parasternalis oder zur Mitte zwischen der letzteren und der Warzenlinie auf dem 6. Rippenknorpel. Die rechte Grenze erstreckt sich vom Brustbeinsrand des 4. Rippenknorpels der linken Seite bis zum Brustbeinsende der 7. Rippe und geht in die untere horizontale Linie über. (Zeichnung *B*.)

C. Gerhardt[1]: Von 12 Fällen bei Kindern im Alter von 3—8 Jahren befindet sich in 11 Fällen der Herzstoss aussen von der linea mamillaris, in 1 Fall auf dieser. Bei der Untersuchung konnte man die Herzschläge im 5. Intercostalraum constatiren. Die verticale Linie

[1] Siehe Th. von Dusch.

des Dreiecks der kleinen Dämpfung beträgt 4.3 Ctm., die horizontale 5 Ctm. Bei mittlerer Länge des Brustbeins 8 Ctm. Der Anfang der kleinen Dämpfung entspricht der Mitte des sternum, in der Hälfte der Fälle dem oberen Rand der 3. Rippe, in $\frac{1}{4}$ der Fälle der 4. Rippe.

E. Barthez et A. Lanné[1]: Die grosse Dämpfung: unten links der Herzstoss oder die 5. Rippe, vertical nach oben vom Herzstoss 4—7 Ctm., rechts der linke Rand des Brustbeins, links die kreisförmige Linie, die von oben von rechts nach links und unten geht. Der Anfang der Dämpfung überragt links die Warze nach aussen, wobei die letztere entspricht der Mitte der äussern Grenze. Im Alter von über 6 Jahren steht die Warze bisweilen etwas höher.

Die Länge des Brustbeins bei Kindern:

3,5—5 Jahre = 11—13 Ctm.
6—10 " = 12—15 Ctm.
11—15 " = 12,5—18 Ctm.

S. O. Wassilewsky[2]: Die Lage des Herzens bei Kindern ist eine mehr horizontale, als bei Erwachsenen, auf Grund des höheren Standes des Zwerchfells. Durch Aufblähen des Bauches kann das Herz um eine ganze Rippe gehoben werden, wobei die Basis desselben nach rechts abweicht, die Spitze nach links.

Der Herzstoss befindet sich 1—2 Ctm. nach aussen von der Warzenlinie, im 4., 5. oder in beiden Intercostalräumen zusammen. Von 1820 Fällen lag er innen von der linea mamillaris in 0,6 %; auf der Linie selbst in 1,5 % und aussen in 98 %. In Hinsicht auf die Intercostalräume lag der Stoss im 4. in 43.3 %, im 4. und 5. in 21.5 % und nur im 5. in 33 %. Nach Daten von 865 Messungen an Lebenden und 112 an Leichen wurden die mittlern Masse der Herzdämpfung, wie folgt, festgestellt: die Breite in der Höhe der Warzen und ebenso die Breite vom äussersten Punkt links bis zum ersten Endpunkt in der Höhe des Zwerchfells. Der erste Querdurchmesser beträgt im Durchschnitt: 7.4 Ctm. im Säuglingsalter; 10.6 im 1.—3. J.; 11.6 im 3.—5. J.; 12.4 im 5.—8. J.; 13.5 im 8.—12. J. Der zweite Querdurchmesser im Säuglingsalter, 7.8 Ctm.; im 1.—3. J., 9.5; im 3.—5. J., 11.5; im 5.—8. J., 12.9; im 8.—12. J., 14.7 Ctm.

Weil[3]: Die kleine Dämpfung des Herzens im Kindesalter fängt höher an und steigt mehr nach links hinunter, als bei Erwachsenen, wobei ihre untere Grenze höher steht, in Zusammenhang mit dem höhern Stand des Zwerchfells. Die Grenzen der kleinen Dämpfung sind im Vergleich mit den Erwachsenen grösser. Die grosse Dämpfung: oben der 3. Intercostalraum, links — in einiger Entfernung vom rechten Rand des sternum. Der linke Rand der grossen Dämpfung geht fast parallel mit dem Rand der kleinen Dämpfung, während der rechte Rand der absoluten Dämpfung vollständig zusammenfällt mit dem linken Rand des Brustbeins. (Zeichnung *C.*)

H. Sahli[4]: Der Herzstoss befindet sich in 18 Fällen bei Kindern im Alter von 9 Mon. bis 12 J. aussen von der linea mamillaris, innen von dieser und auf ihr je 4mal, im 5. Intercostalraum 10mal, im 4., 5mal, in beiden zusammen 3mal; wobei weder das Alter noch die Körperlänge einen bemerkenswerthen Einfluss hatte. Das Erhabensein der Herzspitze bei Kindern kann man nicht erklären durch den hohen Zwerchfellstand, da dabei auch erhoben sein müsste die Basis des Herzens.

Von 25 Fällen befand sich der obere Rand der grossen Dämpfung links: (im Alter von 2, 3 u. 6 J.) 7mal, im 2. Intercostalraum; 3mal (Kinder der Brustperiode, von 6 und 12 J.) auf der 2. Rippe; 2mal auf dem obern Rand der 2. Rippe (Kinder von

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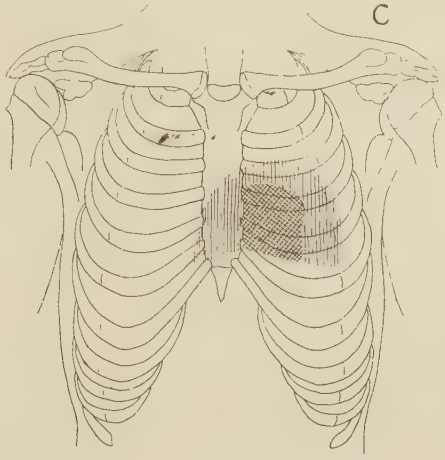
[1] *L. c.*

[2] " Lage und Grenzen des Herzens bei Kindern." *Wratsch*, 1885, No. xxxiii—xxxiv.

[3] *Handbuch und Atlas der topographischen Percussion*, 2. Aufl., Leipzig, 1880.

[4] H. Sahli: *Die topographische Percussion bei Kindern.* Aus dem Deutschen übersetzt. St. Petersburg, 1887, Seite 18—21; 86—107; 117—120.

12 J.); 4mal auf dem untern Rand derselben (Alter von 4, 6, 12 J.) und in 9 Fällen auf dem obern Rand der 3. Rippe (1, 4, 6, 7, 8 u. 10 J.). Die rechte Grenze der grossen Dämpfung befand sich auf der linea parasternalis, in einer Entfernung von der Hälfte der Brustbeinbreite 11mal (im Alter von 9 Mon., 2, 2,5, 4, 6 u. 8 J.), von der ganzen Breite des Brustbeins 6mal (im Alter von 10 Mon., 2, 6, 7 u. 8 J.) Innen von der linea parasternalis ist die erwähnte Grenze gefunden in 6 Fällen (Alter von 3, 10, 12 J.); hiebei war der Abstand derselben von dem rechten Rand des Brustbeins zweimal gleich der Breite der letzteren (10 u. 12 J.). Die linke Grenze der verhältnissmässigen Dämpfung über-



ragte die Warzenlinie aussen um 0.5—2 Ctm. in 21 Fällen, war auf der linea mamillaris nur in 2 Fällen. Eine besondere Abhängigkeit vom Alter war nicht zu finden, wenn auch das Ueberragen nach aussen um 0.5 Ctm. vor dem 6. J. nicht beobachtet wurde.

Der Querdurchmesser der grossen Dämpfung, gerechnet von der Mittellinie und in der Höhe der Warze war links 6—9 Ctm., rechts 2—6 Ctm. und hierbei befand er sich in keiner Abhängigkeit vom Alter. Die ganze Breite der relativen Dämpfung betrug 8.5—14 Ctm.

Im Verhältniss zum Wuchs des Körpers war die Breite der Herzdämpfung in den ersten 3 J. des Lebens fast gleich, ausgedrückt durch 1 : 7,6. Im 4. u. 6. J. sind die Masse der Dämpfung verhältnissmässig zum Wuchs bedeutender, 1 : 8,6, im 7.—12. J. wird das Verhältniss wieder beinahe dasselbe, 1 : 10,7. Im Ganzen zeigt die grosse Dämpfung die Verhältnisse wie bei Erwachsenen, wobei ein besonderer Unterschied nach dem Alter nicht besteht, obgleich bei Kindern die relative Dämpfung grösser ist in allen drei Richtungen nach oben, nach rechts und nach links.

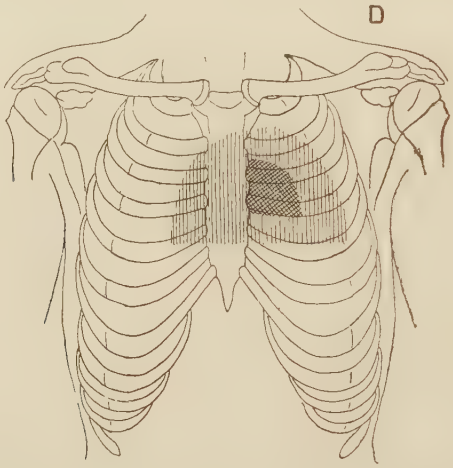
Die individuellen Schwankungen bei Kindern spielen eine grosse Rolle, insofern man im Alter von 12 J. gelegentlich characteristische Verhältnisse finden kann und andererseits können die dem Kindesalter entsprechenden Eigentümlichkeiten abwesend sein bei einem 16jährigen Kinde.

Die kleine Dämpfung fängt oben an vom obern oder untern Rand der 3. Rippe. Ein Unterschied in Bezug auf das Alter ist nicht gefunden. Die Breite der kleinen Dämpfung ist fast gleich in verschiedenen Lebensperioden, und darum ist sie relativ grösser bei kleinen Kindern und im Allgemeinen bedeutender im Kindesalter, als bei Erwachsenen. Die rechte Grenze geht längs dem linken Rande des Brustbeines, die linke geht näher an die Warzenlinie heran, sich bald auf der Parasternallinie, bald zwischen ihr und der Mamillarlinie, zuweilen auf der linea mamillaris sich befindend. Von 23 Fällen bei Kindern im Alter von 11 Tagen bis 12 J. incl., befindet sich der obere Rand der kleinen Dämpfung am obern Rand der 3. Rippe 3mal (Alter von 11 Tagen, 4,5—11 J.), am untern Rand 4mal (9 Mon.,

6, 8 u. 12 J.), auf der Rippe 1mal (12 J.), im 3. Intercostalraum 10mal (2,3 4, 6, 7, 8, 10 u. 12 J.), auf dem oberen Rand der 4. Rippe 5mal (10 Mon., 2,5, 3, 4, 6 J.). In Warzenhöhe: zwischen linea parasternalis und mamillaris in 12 Fällen (11 Tage, 9 Mon., 2,5, 4, 6, 10, 12 J.), auf der linea parasternalis in 9 Fällen (10 Mon., 2, 4, 5, 6, 7, 8, 10 u. 11 J.) und innen von der linea mamillaris in 2 Fällen (2 J.).

Die Breite der kleinen Dämpfung von der Mitte des Brustbeins betrug 2—3 Ctm. im Säuglingsalter und 3.5—5.5 in allen andern Perioden des Kindesalters, wobei weder das Alter noch die Körperlänge einen bemerkenswerthen Einfluss hatten.

Die Breite des Brustbeins ist im Verlauf des ersten Jahres beständig gleich 1—2 Ctm.



und im Durchschnitt 1.3 Ctm. In den andern Perioden schwankt der Querdurchmesser des sternum zwischen 1.4 und 3 Ctm., im Durchschnitt 1.9 Ctm. Die Breite in der Höhe des 5. Intercostalraumes im Vergleich mit derselben in der Höhe des 1. und 2. etwas weniger. Eine besondere Abhängigkeit des Querdurchmessers des sternum von der Länge des Körpers und des Alters ist nicht bemerkbar.

Die Länge des Brustbeins im Alter von 17 Mon. ist 7 Ctm., im 14. J. 14.5 Ctm. Beim 5jährigen Kind ist sie geringer als beim 3jährigen (9 Ctm.), im 2.5 J. grösser als im 10. J. (10.5 Ctm. im ersten Fall, 10 Ctm. im zweiten).

Die linke Warze ist von der linea mediana im ersten Jahr 4—5 Ctm. entfernt, im 2. J. um 5—6 Ctm., im 3. u. 4. J. um 5.5—6.5 Ctm., im 5, 6, 7, 8 J. um 6—7 Ctm. und im Laufe des 9. u. 10. Jahres um 6.5—8 Ctm. In 17 Fällen (Alter von 11 Mon.—11 J.) lag die Warze auf der 4. Rippe 9mal im 4. Intercostalraum 5mal und im 3. Intercostalraum 3mal. (Zeichnung *D.*)

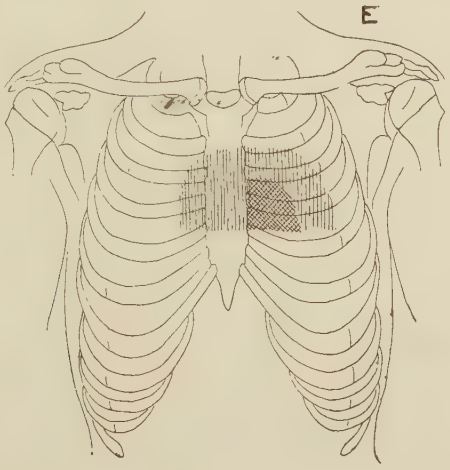
W. von Starck[1]: In den ersten Lebensjahren nimmt das Herz bei Kindern mehr Raum ein, wegen der relativ geringeren Breite des Brustkastens. Der Herzstoss befindet sich in den ersten 3 Lebensjahren in der Mehrzahl der Fälle aussen von der Warzenlinie, in den darauf folgenden Jahren wird solch eine Erscheinung immer seltener beobachtet, und vom 13. Jahre an fast nie. Gerade auf der linea mamillaris befindet sich der Herzstoss bei Kindern im Säuglingsalter selten, und nachher bis zum 7. Jahr immer öfter. Nach dem 7. Jahr constatirt man den Herzstoss auf der Warzenlinie wieder seltener, obgleich im 14. Jahre seine Lage an dieser Stelle eine gewöhnliche Erscheinung ist.

Innen von der Mamillarlinie wird der Herzstoss bis zum Alter von 2 J. nicht beobachtet,

[1] "Die Lage des Spitzenstosses und die Percussion des Herzens im Kindesalter." (*Archiv für Kinderheilkunde*, Bd. ix, S. 241—293. 1888.)

im Laufe des 3. bis 6. J. kommt er vor, aber selten; vom 9. J. an — in der Mehrzahl der Fälle, und endlich vom 13. J. an fast immer.

In Hinsicht auf die Intercostalräume pflegt der Herzstoss im Säuglingsalter fast ausschliesslich in der Höhe des 4. sich zu finden. Entsprechend dem Wachsthum wird das Finden des Stosses im angegebenen Raum immer seltener. In den ersten 2 Jahren ist der Stoss in seltenen Fällen gleichzeitig im 4. und 5. Raum zu finden, im 3. bis 6. Jahr ist diese Erscheinung häufig, aber darauf wieder selten. Im 5. Raum allein findet man ihn sehr selten bei Kindern der ersten 2 Lebensjahre, in den folgenden Jahren öfters, vom 7. J. an — meistenteils, und vom 13. J. an — fast stets. Was den 6. Raum betrifft, so muss man das Vorhan-



densein des Stosses als äusserst seltene Erscheinung ansehen. Im Allgemeinen kann man folgende Behauptung als annehmbar erklären: In der ersten Kindheit liegt der Spitzenstoss aussen von der Mamillarlinie, im mittleren Kindesalter auf dieser, im späteren Kindesalter innen von derselben. In der frühen Kindheit geht der Stoss hinunter um 2 Ctm. von der Mamillarlinie, in späterer — um 1 Ctm.

Auf Grundlage von 300 Fällen ganz gesunder Kinder kann man 3 Typen der grossen Herzdämpfung feststellen: Die Säuglingsperiode, das 6. Jahr und das 12. Jahr.

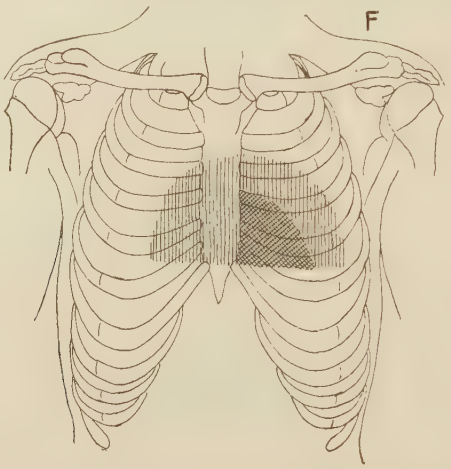
Der erste Typus. Oben links das Ende der zweiten Rippe am Brustbein, seltener der 2. Intercostalraum in geringem Abstand vom Brustbein auf demselben; oben rechts die völlige Abwesenheit der Dämpfung oder das Bild, welches der linken Seite eigen ist; vom obern Punkt geht links die Grenze der Dämpfung kreisförmig nach unten, auf ihrem Weg die 3. Rippe schneidend auf der Warzenlinie oder innen von derselben, die 4. Rippe schneidend um 1—1.5 Ctm. aussen von der Mamillarlinie und die 5. Rippe um 2—2.5 Ctm. von dem obern Punkt rechts in der Nähe der rechten Parasternallinie, oft auf ihr selbst nach unten zum 4. rechten Intercostalraum oder zum oberen Rand der 5. Rippe. Die Grenze rechts steht am weitesten ab vom Brustbein in der Höhe der Warze. Die untere Grenze ist horizontal und geht längs dem obern Rand der 5. Rippe. (Zeichnung *E*.)

Die Breite der grossen Dämpfung in Warzenhöhe 6.6—8 Ctm., wobei links 5.1—5.6 Ctm., rechts 1.6—2 Ctm.

Die kleine Dämpfung: oben der untere Rand der 3. Rippe links, nach unten der linke Brustbeinrand, nach unten und aussen die horizontale Linie der grossen Dämpfung, nicht weit von der Mamillarlinie. Die Höhe der kleinen Dämpfung im Durchschnitt 3 Ctm., breit 3.4 Ctm. (bei einer Breite des Brustbeins von 6 Ctm.).

Der zweite Typus. Die grosse Dämpfung: oben und links der 2. Intercostalraum, die

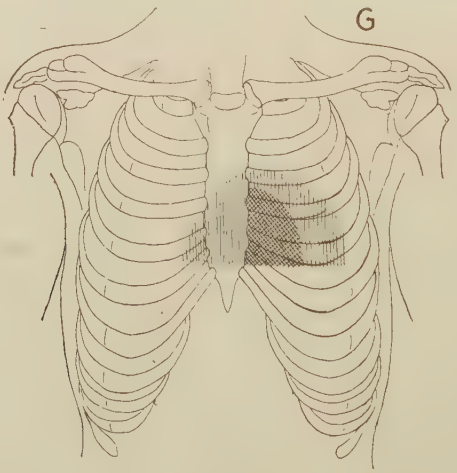
linke Grenze schneidet die 3. Rippe in der Parasternallinie oder etwas aussen von dieser, die 4. Rippe in der Mamillarlinie oder etwas aussen die 5. um 1—1.5 Ctm. hinter der Mamillarlinie ; in der Mitte des 5. Intercostalraumes vereinigt sich diese Grenze mit der unteren hori-



zontalen. Rechts ist die Grenze der Dämpfung mehr ausgebogen, als beim ersten Typus und ist weniger vertical ; sie geht am untern Rand der 5. Rippe rechts in die untere horizontale Linie über.

Die Breite der grossen Dämpfung ist 10.2 Ctm., wovon auf die rechte Seite 2.9 Ctm. kommen, auf die linke 7.3. Die linke Grenze geht hinaus über die Stelle des Herzstosses um 1—2 Ctm. Die kleine Dämpfung: oben der obere Rand der 4. Rippe. Die Höhe 3.6 Ctm., die Breite 4 Ctm. (die Länge des Brustbeins 9 Ctm.). (Zeichnung *F*.)

Der dritte Typus. Die grosse Dämpfung: oben und links das zum Brustbein hin liegende



Ende der 3. linken Rippe ; die linke Grenze geht durch den untern Rand der 3. linken Rippe auf der Parasternallinie, durch die 4. Rippe nach innen von der Mamillarlinie, schneidet die 5. auf der Mamillarlinie oder geht aussen von dieser und erreicht unten den untern Rand der 6. linken Rippe. Rechts ragt die Grenze über den rechten Rand des Brustbeins hervor bis zur Befestigungsstelle der 4. rechten Rippe, geht nach unten ganz parallel dem rechten Rand

des Brustbeins, zwischen der Linea sternalis und parasternalis und geht im 5. rechten Intercostalraum in die untere horizontale über. Die linke Grenze geht über die Stelle des Herzstosses hinüber um 1—1.5 Ctm. nach aussen.

Die Breite der grossen Dämpfung ist 11.5 Ctm., wobei 3.3 Ctm. auf die rechte Seite kommen und 8.2 auf die linke.

Die kleine Dämpfung ist, wie bei Erwachsenen: oben der untere Rand der 4. linken Rippe, die Höhe 4.3 Ctm. und die Breite 4.2 Ctm. (Zeichnung *G.*)

Der Abstand zwischen der linken Warze und der Mittellinie vergrössert sich mit dem Alter stetig und recht regelrecht. Man bemerkt keinerlei Stillstände und Pausen im Wachsthum des genannten Raumes.

Die mittlere Länge des Brustbeins im 1. Lebensjahr ist 6.4 Ctm., vom 1. bis 4. J. 7.4 Ctm., 5. bis 7. J. 9 Ctm., 8. bis 12. J. 11.3 Ctm.

I. Keating [1] und W. Edwards: Das Herz bei Kindern liegt höher als bei Erwachsenen. Die höhere Lage seiner Spitze kann abhängen von der Erweiterung des Darmes und der relativen Grösse der Leber.

Der Herzstoss fällt in einer Reihe von Fällen mit der linken Warze zusammen im 4. Intercostalraum. Auf Grund von 96 Fällen geht hervor, dass die Herzspitze um so höher steht, als das Alter geringer ist: im 6. J. auf der Warze, im 8. J. um 1.5—2.5 Ctm. tiefer.

Die grosse Dämpfung: links unten die Warze und Vereinigung des Brustbeinkörpers mit dem processus xyphoideus, oben und links der 3. Rippenknorpel.

Die kleine Dämpfung beginnt oben mit dem 4. Rippenknorpel der linken Seite oder dem 3. Intercostalraum.

W. F. Jacubowitsch [2]: Viele Autoren meinen, dass das Herz bei Kindern um so horizontaler liegt, je geringer das Alter ist, dass der Herzstoss aussen von der Warzenlinie liege, und die Herzschläge im 4. Intercostalraum zu fühlen sind. Diese Behauptungen kann man nicht für die unbestreitbare Wahrheit halten, da die Topographie des Herzens noch nicht als vollendet von klinischen Seiten zu betrachten ist.

Der höhere Zwerchfellstand spricht noch nicht für die höhere Lage des Herzens, da für Bestimmung dieser Lage man genau die obere Grenze in Bezug auf die Knochentheile feststellen muss.

Der Herzstoss liegt aussen von der Mamillarlinie nicht mehr als 1 Ctm. und dann im 5. Intercostalraum. Die Grenzen der grossen Dämpfung: links und oben die Stelle der Vereinigung mit dem Brustbeinknorpel der 2. Rippe oder der 2. Intercostalraum, rechts und oben der 2. Intercostalraum oder der 3. Rippenknorpel, unten und links der untere Rand der 6. linken Rippe, unten und rechts das Brustende des 6. rechten Rippenknorpels. Die rechte Seitengrenze steht vom Brustbein, im obern Theil um halb so viel ab, wie dessen Körper beträgt, im untern Theil fällt sie mit der linea sternalis dextra zusammen.

Die kleine Dämpfung: links und oben das Brustende des 4. Rippenknorpels, links und unten das Brustende des 7. linken Rippenknorpels. Links geht das Gebiet der Dämpfung vom 4. Rippenknorpel kreisförmig bis zur Parasternallinie oder bis zur Mitte der Entfernung zwischen ihr und der Mamillarlinie, endend auf der 6. Rippe.

G. MacClellan [3]: Der Herzstoss bei Kindern liegt auf der Warzenlinie oder innen von dieser. Bei kleinen Kindern liegt die Spitze im 4. Intercostalraum. Eine höhere Lage des kindlichen Herzens steht in Verbindung mit der hohen Lage des Zwerchfells und der mehr schrägen Lage der Rippen.

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[1] *Diseases of the Heart and Circulation in Infancy and Adolescence.* Philadelphia and London, 1889, pp. 10, 13, 17.

[2] *Handbuch für die Diagnostik der Kinderkrankheiten und die Methode der Untersuchung der Kinder.* St. Petersburg, 1890, S. 195–200.

[3] "On the Anatomy of Children." *Cyclopædia of the Diseases of Children,* by M. Keating, Vol. i., 1889, pp. 28–29.

I. W. Ballantyne [1]: Das Herz der Kinder erscheint grösser wegen der geringeren Breite des Brustkastens. Es liegt mehr quer, und darum auch höher im Verhältniss zur Wirbelsäule. Die Spitze liegt bald in der Mamillarlinie, bald innen oder aussen von dieser.

Alfr. Vogel,[2] Ph. Biedert: Die obere Grenze der grossen Dämpfung fängt links auf der 3. Rippe an; die Breite derselben erstreckt sich von der Mitte oder dem linken Rand des Brustbeins bis zur linken Mamillarlinie. Der Herzstoss befindet sich auf der Warzenlinie, im 4. oder 5. Intercostalraum. Das Herz in toto liegt bei Kindern höher als bei Erwachsenen.

H. B. Whitney [3]: Bis zum Beginn des 6. Jahres zeigt die grosse Dämpfung bei Kindern dieselben Grenzen, wie bei Erwachsenen. Die nach links ausgebogene Linie geht von der Vereinigungsstelle der 3. Rippe mit der Brustlinie nach aussen und unten in der Richtung zur Herzspitze, im 4. oder 5. Intercostalraum, nahe an der Mamillarlinie vorbei. Rechts zieht die Dämpfungsgrenze perpendiculär auf dem linken Rand des Brustbeins. In den ersten 5 Lebensjahren ist der Percussionston ganz gleich in der obern und untern Hälfte des Brustbeins. Vom 6. Jahre an kann man schon eine bedeutende Dämpfung erkennen auf der untern Hälfte desselben. In den meisten Fällen haben die 5—9jährigen Kinder dieselben Grenzen der Dämpfung wie die Erwachsenen und die Kinder in den 4 ersten Lebensjahren. Beginnend vom 5. Jahr, wo in allen Fällen ohne Ausnahme die Dämpfung den Character der "infantile præcordia" trägt, kommt immer häufiger das "enlarged præcordia" vor, die dann als beständige Erscheinung bleibt. Die Grenze der Dämpfung ändert sich in dem Sinne, dass links und oben sie auf den 2. Intercostalraum fällt, ja sogar auf die 2. Rippe, links und unten um 0.75—1.5 Ctm. nach aussen von der Mamillarlinie, rechts um 3—4 Ctm. über dem rechten Rand des Brustbeins hinausgehend. In der Richtung nach oben nähert sich die rechte Grenze allmählich dem Brustbein.

E. Weil [4]: Die Spitze des Herzens befindet sich grösstentheils im Kindesalter im 4. Intercostalraum.

W. Uffelmann, B. Bendix [5]: Bei Kindern zwischen 6 und 12 Monaten geht die Linie der Herzdämpfung rechts nicht weiter als bis zum linken Rand des Brustbeins, wobei der Umfang derselben um so grösser ist, je geringer das Alter.

Die Grenzen der grossen Dämpfung: links und oben der obere Rand der 3. Rippe in der Nähe des Brustbeins. Rechts zieht die Grenze längs dem linken Rand des sternum bis zum untern Rand der 5. Rippe, horizontal nach links bis zur Mamillarlinie, von da nach oben längs derselben Linie bis zum obern Rand der 4. Rippe, und dann in schräger Richtung nach oben und rechts.

A. Edwards [6]: Die Warze liegt gewöhnlich im 4. Intercostalraum oder auf der 4. Rippe, kann aber auch auf dem 3. und 5. Raum liegen. In allen Epochen des Kindesalters geht die Herzdämpfung über den rechten Rand des Brustbeins hinaus.

Die Lage des Herzens ist eine höhere, manchmal sogar fast horizontale.

Die Spitze liegt in der ersten Lebenszeit auf der Mamillarlinie, im nachfolgenden Alter nach aussen oder innen von ihr und am häufigsten im 4. bis 5. Intercostalraum.

Im Anfang seines Artikels bemerkt der Autor ganz mit Recht, dass es in der Medicin wenig Fragen gibt, die so arm an gelehrtem Material und so reich an sich widersprechenden Meinungen sind.

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[1] *An Introduction to the Diseases of Infancy*, 1891, Edinburgh, pp. 65–66, 75.

[2] *Lehrbuch der Kinderkrankheiten.* Uebersetzt aus dem Deutschen, 1891, S. 17.

[3] "The Normal Præcordia in Childhood" (*Archives of Pediatrics*, 1894, pp. 802–807). "The Præcordial Area in Children" (*Journal of the American Medical Association*, 1898, Vol. xxxi., No. 19).

[4] "Maladies acquises de l'appareil circulatoire." *Traité des maladies de l'enfance*, par I. Groucher, J. Comby et A. Marfan, T. iii., Paris, 1897.

[5] *Lehrbuch der Kinderheilkunde*, 2. Aufl., Berlin und Wien, 1899, S. 294–295.

[6] "The Normal Præcordia of Infancy and Childhood" (*Cyclopædia of the Diseases of Children.* Vol. v. Supplement. Philad., 1899, pp. 560–567).

Wir werden uns nun bemühen, einen Schluss aus allen litterarischen Daten zu ziehen und hernach die Resultate eigener Beobachtungen der geneigten Beachtung seitens der Collegen zu unterstellen.

a. Die von allen Autoren anerkannte mehr horizontale und hohe Lage des Herzens bei Kindern steht in geradem Verhältniss zum relativ hohen Stand des Zwerchfells, bei mehr schräger Richtung der Rippen, wegen Ueberwiegen des Längsdurchmessers des Brustbeins über den Querdurchmesser und der relativen Grösse der Leber.

Die Aufblähung des Magens und Darms kann die Rolle eines ætiologischen Momentes nur in den Fällen spielen, die über die Grenzen des normalen hinausgehen.

Der höhere Stand der Herzensbasis ist kaum unumgänglich nötig, wenn man die Abweichung des oberen Teils über den rechten Rand des Brustbeins hinaus in Betracht zieht.

b. Der Herzstoss kann nicht als hinweisend für die Bestimmung der Dämpfungsgrenzen dienen, weil seine Lage nicht genügend bestimmt ist und man ihn mit grosser Mühe nur in den ersten Lebensjahren constatiren kann. Richtig ist nur eins, dass der Stoss bei kleinen Kindern fast immer aussen von der Mamillarlinie liegt, in der zweiten Kindheit auf dieser. In Bezug auf die Intercostalräume nimmt der Herzstoss eine recht unbestimmte Lage ein, wenngleich er bei kleinen Kindern vorherrschend, fast ausschliesslich im 4., bei älteren im 5. Intercostalraum sich befindet. Gar nicht selten ist der Stoss deutlich in beiden Intercostalräumen gleichzeitig bemerkbar.

c. In verticaler Richtung kann die Warze in ihrer Lage wechseln zwischen der 3. und 5. Rippe, in der Mehrzahl der Fälle liegt sie auf der 4. Der Raum zwischen der Mamillarlinie und der linea mediana sterni vergrössert sich dem Alter entsprechend beständig und gleichmässig, von 4.5 Ctm. im Säuglingsalter bis zu 7.5 Ctm. im 10. Jahre, auf Grund wessen die bezeichneten Linien als völlig zuverlässige Hülfsmittel bei der Untersuchung der Grenzen der Herzdämpfung dienen können.

d. Die Spitze des Herzens fällt nicht zusammen mit dessen Stoss, indem dieselbe sich bei kleinen Kindern an der Stelle der Warze oder in deren Nähe befindet, bei grössern Kindern um 1.5 — 2.5 Ctm. tiefer und nach aussen von der Mamillarlinie um 1—2 Ctm. In den ersten Lebensjahren befindet sich die Spitze im 4. Intercostalraum, in der Mehrzahl der Fälle im 5., sehr selten im 6. Intercostalraum.

e. Die Grenze der grossen Herzdämpfung zeigt sich in folgender Gestalt. Links und oben: der 2. Intercostalraum, der obere Rand der 2. Rippe, diese Rippe selbst, ihr unterer Rand, der obere Rand der

3. Rippe und die letztere selbst, wobei, beginnend vom Säuglingsalter, der obere Rand der Dämpfung beständig sich senkt. In der Nähe des Brustbeins erreicht der Raum der Dämpfung 0.5—1.5 Ctm. Nach den Daten von H. Sahli sind die Lage der obern linken Grenze und das Alter der Kinder nicht gegenseitig von einander abhängig. Rechts und oben: die 2. rechte Rippe, der 2. Intercostalraum oder die 3. Rippe. Es ist möglich ein Ueberragen über den Rand des Brustbeins um 0.5—1 Ctm. Links und seitlich: die kreisförmige Linie, die von der obern linken Grenze kommt und die 3. Rippe auf der Parasternal- oder Mamillarlinie schneidet und auch etwas aussen von ihnen, die 4. Rippe auf der Mamillarlinie oder um 1—1.5 Ctm. nach aussen, zuweilen nach innen von dieser, die 5. Rippe auf der Mamillarlinie oder um 0.6—2.5 Ctm. ausserhalb derselben. Die genannte Linie geht dann über in die untere horizontale Linie in der Gegend des 4. oder 5. Intercostalraumes in der frühen Kindheit oder auf dem obern, sogar untern Rand der 6. Rippe im spätern Kindesalter, nach aussen um 1—2 Ctm. von der linea mamillaris sinistra abweichend. Links und unten: aussen vom Herzstoss, oft auf der Mamillarlinie, ausserhalb derselben, um 1—1.5 Ctm., nach dem 6. Jahr sogar um 2 Ctm. Längs der Mamillarlinie begiebt sich die Seitengrenze links von unten nach oben in einer Ausdehnung von 4—7 Ctm., bis zum oberen Rand der 4. linken Rippe, wobei die Warze gewöhnlich die Mitte der linken äussern Grenze der grossen Herzdämpfung einnimmt. Rechts und seitlich: von der 2. rechten Rippe, in Form einer etwas ausgebogenen Linie bis zur 6. Rippe oder bis zum 6. Intercostalraum, in der Nähe des rechten Brustbeinrandes. Diese Grenze kann bis zum linken Rand des Brustbeins gehen, vorherrschend bei Kindern der ersten 4 Jahre, wobei im spätern Alter dieselbe nach rechts sich begeben kann um die Hälfte der Breite des sternum oder um 1—4 Ctm. von der linea mediana. Der bei Kindern, welche das Säuglingsalter hinter sich haben über den rechten Rand des Brustbeins gewöhnlich hinüberragende Teil der grossen Herzdämpfung nimmt den Raum der 3., 4., und 5. rechten Rippen ein, mit dem grössten Diameter in der Höhe der Warze und anfänglich der Parasternallinie entsprechend, ja weiter gehend, und darauf der rechten Sternallinie. Unten: die Horizontallinie von der 6. Rippe oder dem Raum rechts, am Brustbeinrand bis zum unteren Rand derselben Rippe oder von dem Raum der linken Seite zum endgültigen Grenzpunkt. In Hinblick auf das Brustbein trifft diese Linie auf die Stelle der Vereinigung des Brustbeinkörpers mit dem Processus Xiphoideus.

f. Die mittlere Breite der grossen Dämpfung in der Höhe der Warzen beträgt im Säuglingsalter 7 Ctm., wobei auf den linken Teil 5 Ctm., auf den rechten 2 Ctm. kommen. Von 1—6 Jahren beträgt sie

10 Ctm., von denen auf die linke Seite 7 Ctm., auf die rechte 3 Ctm. kommen. Im 6. bis 12. J. ist die Breite im Allgemeinen 12 Ctm., 8.5 links und 3.5 rechts. Die mittlere Länge des Längsdurchmessers, gerechnet von der Spitze des Herzens bis zum rechten Endpunkt unterscheidet sich bei Kindern jeden Alters fast gar nicht von dem Querdurchmesser, was ganz den Folgerungen von A. Steffen's widerspricht, nach welchem zwischen beiden Diametern ein Verhältniss von 2 zu 3 besteht.

Im Verhältniss zum Wuchs des Körpers pflegt die Breite der grossen Dämpfung am bedeutendsten in der dritten Periode, am geringsten in der ersten zu sein.

g. Die kleine Herzdämpfung hat die Form eines fast regelmässigen geradwinkligen Dreiecks, dessen Verticalkathete gewöhnlich grösser als die horizontale ist. Die Grenzen derselben sind folgende:

Oben: Im 1. Lebensjahr, der obere oder untere Rand der 3. Rippe, an der Stelle der Vereinigung dieser mit dem Brustbein, in der 2. Kindheitsperiode der 3. Intercostalraum, danach der obere, sogar untere Rand der 4. Rippe. Rechts und unten: die Verticallinie, die längs dem linken Rand des Brustbeins nach unten bis zur Vereinigung mit der horizontalen Grenze der grossen Dämpfung geht, im 6. Intercostalraum oder auf der 7. Rippe, an deren Anheftungsstelle am Brustbein. Unten: der innere Theil der Horizontallinie, gerechnet von dem Brustbein bis zur Mitte zwischen der linea parasternalis und mamillaris, selten auf der erstern von beiden und noch seltener in der Nähe der zweiten. Von rechts nach links — die Hypothenuse, welche die Enden beider Katheten vereinigt.

h. Die verticale Kathete des Dreiecks der kleinen Dämpfung im Durchschnitt für alle Kindheitsperioden beträgt 3.5 Ctm., mit Schwankungen nach einer oder der anderen Seite um 1 Ctm., die horizontale 4 Ctm., mit fast denselben Schwankungen.

i. Die Länge des Brustbeins beträgt durchschnittlich für jedes Alter 10 Ctm., wobei sie bei Brustkindern 6.5 Ctm. erreicht, im 2.—6. J. 1½mal mehr, nach dem 10. Jahr 2mal mehr. Die mittlere Breite des Körpers des Brustbeins in allen Kindheitsperioden beträgt 1.6 Ctm., wobei im Säuglingsalter dieselbe nicht mehr sein kann als 1 Ctm., und am Anfang der Geschlechtsreife 3 Ctm. erreicht.

Bei einer durchschnittlichen Länge des Brustbeins von 8 Ctm., beginnt nach C. Gerhardt die kleine Dämpfung gerade in der Mitte der Entfernung des Heftes von der Stelle der Anheftung des Processus Xyphoideus.

Indem wir uns an die Auseinandersetzung der eigenen Daten machen, kann ich nicht mit Stillschweigen übergehen die in letzter

Zeit von O. Hauser[1] hervorgehobene Frage von der äussersten Schwierigkeit, ja fast Unmöglichkeit der Bestimmung der Herzgrenzen bei kleinen Kindern, mit Inhilfenahme der Percussion und Palpation, besonders der Linie der grossen Dämpfung. Indem der geschätzte Autor auf die Methode der Untersuchung durch die Röntgenstrahlen als auf die genaueste hinweist, lässt er die Möglichkeit einer schädlichen Wirkung der X-Strahlen auf das Gebiet des allerwesentlichsten Organs in der Lebensökonomie ausser Acht. Andrerseits, mit Geduld, bei Erfahrung und strenger Beobachtung der Regel, dass man zum Zweck der Bestimmung der Dämpfungsgrenzen ausschliesslich die Fälle benützt, in welchen pathologische Abweichungen nicht vorhanden sind, kann man immer das gewünschte Resultat mit Hülfe der gewöhnlichen Untersuchungsmethoden erlangen.

Als Material für die eigenen Daten dienten nur die Kinder des Sommersanatoriums und die Ambulanz der Heilanstalt der Gesellschaft der Hilfsleistung für kranke Kinder. Bei der Auswahl der Fälle wurden streng die Regeln beobachtet, dass untersucht wurden nur diejenigen Kinder, welche keine Abweichung von normaler Entwickelung und auch keine pathologischen Störungen zeigten, welche in irgend welcher Art auf die Veränderung der Dämpfungsgrenzen hinwirken könnten. Im Voraus wurde die Bestimmung der Körperlänge gemacht, diejenige des Brustumfanges in der Höhe der Warzen und des Körpergewichtes. Dann auf dem Wege einer vorsichtigen wiederholten Percussion und Palpation wurden die äussersten Punkte der grossen und kleinen Herzdämpfung bestimmt, mit darauf folgender Vereinigung derselben durch Linien. So sehr auch das Klopfen als ganz zuverlässiges und richtiges Mittel zur Bestimmung der kleinen Dämpfung sich erweist, so muss man mit dieser Methode durchaus vorsichtig sein bei der Untersuchung der Grenzen der grossen Dämpfung, wenn die Palpation genauere Resultate ergiebt.

An der Hand der Gesetze des Körperwachsthums, nach denen das Gewicht am Ende des 1. Jahres sich verdoppelt im 6. Jahre, das Gewicht im 13. Jahre zweimal so gross ist, als im 6. Jahre, habe ich die von mir beobachteten Fälle (229) in 3 Gruppen getheilt, von denen auf die erste, das Säuglingsalter oder *die erste Kindheitsperiode* (33 Fälle), auf die zweite, die Periode vom 2. bis 6. Jahre, oder *die zweite Kindheitsperiode* (46 Fälle), und auf die dritte, die Periode vom 7. bis 12. Jahre, oder *die dritte Kindheitsperiode* (150 Fälle) fällt. Knaben waren mehr, als Mädchen; hierbei führte die Verschiedenheit des Geschlechts keinerlei wesentliche Abweichung mit sich.

[1] "Ueber scheinbare idiopathische Herzvergrösserung bei Kindern." *Centralblatt für Kinderheilkunde*, No. x., 1899.

Der Herzstoss entsprach nicht der Spitze und befindet sich in der ersten Periode in der Warzenlinie, in der zweiten und dritten innerhalb derselben. In Bezug auf die Intercostalräume ist von mir der Stoss fast stets im 4. bemerkt worden, und wenn der Stoss auch im 5. Intercostalraum bemerkt wurde, so war er gleichzeitig auch im 4. zu constatiren, wenn auch sein Vorhandensein nicht so deutlich erkennbar war. Bei Kindern der Säuglingsperiode befand sich der Stoss in allen Fällen ausnahmslos im 4. Intercostalraum, in der zweiten Periode in 11 von 46 Fällen war der Stoss bemerkbar oder fühlbar auch gleichzeitig im 5. Intercostalraum. Von 146 Fällen der dritten Periode war der Stoss in 114 Fällen im 4. Intercostalraum, im 4. und 5. in 35 Fällen, wobei bei Kindern älter als 10 Jahre der 5. Intercostalraum wirklich deutlichere Anzeichen gab.

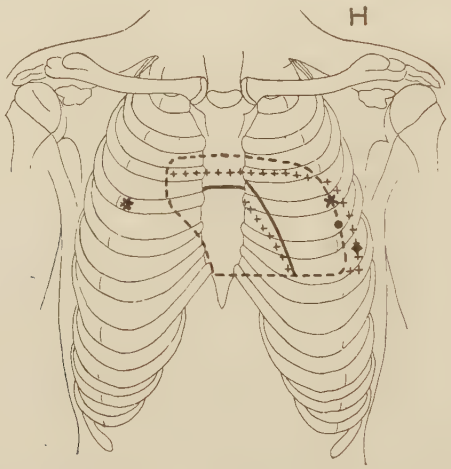
Die Spitze des Herzens liegt in der ersten Periode gewöhnlich im 4. Intercostalraum, dann auch im 5., wobei die untere linke Grenze der grossen Dämpfung bei Kindern im Säuglingsalter auf der Warzenlinie anfängt, um 1.75 Ctm. (im Durchschnitt) unter der Warze (minimum 1 Ctm., maximum 2.5 Ctm.). Im Alter vom 2. bis 6. Jahre incl. fällt der untere linke Grenzpunkt in 36 Fällen von 46 auf die Warzenlinie, und in 10 Fällen von 0.5—1.5 Ctm. ausserhalb derselben, aber immer unter die Warze, durchschnittlich um 2 Ctm. (minimum 1 Ctm., maximum 3 Ctm.). Im Laufe der dritten Periode war die Spitze des Herzens nur in 8 von 150 Fällen, innerhalb jedoch um ebensoviele Ctm. in 12 Fällen. Unter der Warze war der äusserste Punkt der Dämpfung im Durchschnitt um 2 Ctm. entfernt (minimum 0.5, maximum 3.5 Ctm.). Die Warze befand sich in den allermeisten Fällen auf der 4. Rippe oder 0.5 Ctm. über oder unter ihr, sehr selten war die Abweichung davon bedeutender — um einen ganzen Intercostalraum. Was den Abstand der Warzenlinie von der Mittellinie des Brustbeins betrifft, so wird er wirklich grösser in entsprechendem Grade, in directer Abhängigkeit von dem regelrecht dem Alter entsprechenden Wachsthum des Brustumfangs. Ausserdem habe ich mich durch eigene Erfahrung davon überzeugt, dass die äussere linke Grenze der grossen Dämpfung längs der Warzenlinie in einer Ausdehnung von 4—6 Ctm. zieht, entsprechend der Vergrösserung des Brustkorbes und dass die Warze annähernd die Mitte der äusseren Grenzlinie einnimmt.

Die obere Grenze der grossen Dämpfung befand sich in der ersten und zweiten Kindheitsperiode im 2. Intercostalraum; auf beiden Seiten des Brustbeins betrug die Oberfläche der Dämpfung durchschnittlich 1 Ctm. (minimum 0.5, maximum 1.5 Ctm.). In der Periode vom 7. bis 12. J. entsprach der Anfang der Dämpfung dem 2. Intercostalraum in 120 von 150 Fällen, wobei sie auf die 3. Rippe und den untern Rand

derselben nur in 30 Fällen fiel, bei Kindern, die das äusserste Ende der dritten Periode der Kindheit erreichten oder erreicht hatten.

Die linke seitliche Grenze geht bogenförmig von rechts oben und innen nach links unten und aussen, völlig entsprechend den Daten, welche andere Autoren gefunden haben. In der Periode der ersten und zweiten Kindheit geht diese Grenze in die untere Horizontallinie über im Gebiet des 4. oder 5. Intercostalraums, bei ältern Kindern, der 6. Rippe.

In allen Perioden des Kindesalters ragt die grosse Dämpfung des Herzens über den rechten Rand des Brustbeines in der Gegend der 2.



und 3. rechten Rippe hinüber. Der weitere Verlauf der rechten Grenzlinie der Dämpfung ist entsprechend der Beschreibung der Autoren. Der von mir in dieser Hinsicht bemerkte Unterschied besteht bloss darin, dass der grösste Durchmesser des Dämpfungsgebietes auf die rechte Seite des Brustbeins, nicht in der Höhe der Warze, sondern auf der Hälfte der Brustbeinlänge fällt. Man kann durchaus nicht als mit der Behauptung einverstanden sich erklären, dass bei Kindern der ersten 4 Jahre des Lebens die rechte Grenze der relativen Dämpfung des Herzens längs dem linken Rande des Brustbeins verläuft (siehe Bild H). Der Durchmesser der grossen Dämpfung in der Höhe der Mitte des Brustbeins während des Säuglingsalters beträgt im Durchschnitt 6 Ctm. (minimum 5, maximum 7 Ctm.), von denen auf die linke Seite, gerechnet von der linea mediana, 4 Ctm. kommen (minimum 3, maximum 5 Ctm.) auf die rechte 2 Ctm. (minimum 1.5, maximum 2.5 Ctm.). In der zweiten Kindheitsperiode ist die mittlere Breite gleich 7.5 Ctm. (minimum 6, maximum 9 Ctm.), hiervon links 5 Ctm.

(minimum 4, maximum 6 Ctm.), rechts 2.5 Ctm. (minimum 2, maximum 3 Ctm.). In der Periode vom 7. bis 12. J. erreicht besagte Breite durchschnittlich 9 Ctm. (minimum 8, maximum 10 Ctm.), wobei links 6 Ctm. (minimum 5, maximum 7 Ctm.) und rechts 3 (minimum 2.5, maximum 3.5 Ctm.).

Die untere (horizontale) Grenze, der grossen Dämpfung, bis zur Mittellinie gerechnet, beträgt in der ersten Periode im Durchschnitt 5 Ctm. (minimum 4, maximum 6 Ctm.), in der zweiten Periode 6 Ctm. (minimum 5, maximum 7 Ctm.), in der dritten Periode 7 Ctm. (minimum 6, maximum 8 Ctm.).

Die kleine Herzdämpfung hat die Form eines rechtwinkligen Dreiecks, dessen verticale Kathete längs dem rechten Rande des Brustbeins hinzieht, die innere Grenze bildend, dessen kleine Kathete einen Theil der Horizontallinie der grossen Dämpfung vorstellt, und dessen Hypothenuse, welche die beiden Enden beider Katheten vereinigt, bietet gleichzeitig die linke äussere Grenze der Herzdämpfung. Der Anfang der kleinen Dämpfung am Brustbein bot, meinen Beobachtungen gemäss, keine wesentlichen Abweichungen von den Daten anderer Autoren, aber die von C. Gerhardt vorgebrachte und in practischer Hinsicht äusserst wichtige Behauptung, wonach der Anfang der verticalen Kathete genau mit der Mitte des Brustbeins zusammenfällt bei bestimmter Länge desselben, ist leider nicht bestätigt worden. Die verticale Kathete der kleinen Dämpfung in der Periode der ersten Kindheit beträgt 3 Ctm. (minimum 2, maximum 4 Ctm.), in der zweiten Periode 4 Ctm. (minimum 3, maximum 5 Ctm.) in der dritten Periode 5 Ctm. (minimum 4, maximum 6 Ctm.).

Die horizontale Kathete oder die horizontale Grenze der kleinen Dämpfung im Säuglingsalter beträgt durchschnittlich 2.5 Ctm. (minimum 2, maximum 3 Ctm.), bei Kindern der zweiten Periode 3.5 Ctm. (minimum 3, maximum 4 Ctm.), bei Kindern der dritten Periode 4.5 Ctm. (minimum 4, maximum 5 Ctm.).

Die mittlere Länge der grossen Dämpfung des Herzens am linken Rande des Brustbeins in der ersten Periode beträgt 5 Ctm. (minimum 4, maximum 6 Ctm.), in der zweiten Periode 6 Ctm. (minimum 5, maximum 7 Ctm.), in der dritten Periode 7 Ctm. (minimum 6, maximum 8 Ctm.).

Die Länge des Brustbeins, gerechnet vom Schaft bis zum Beginn des Processus Xyphoideus beträgt bei Kindern des Säuglingsalters durchschnittlich 7 Ctm. (minimum 6, maximum 8 Ctm.), in der zweiten Periode 9 Ctm. (minimum 7, maximum 11 Ctm.), in der dritten Periode 11 Ctm. (minimum 9, maximum 13 Ctm.).

Die Entfernung vom manubrium sterni bis zum Anfang der grossen

Dämpfung des Herzens am Brustbein in der ersten Periode betrug durchschnittlich 2 Ctm., in der zweiten Periode 3 Ctm., in der dritten Periode 4 Ctm.

Dass das Herz, während allen Perioden des kindlichen Alters, im Brustraum mehr Platz einnimmt, als im Organismus, welcher am Ende seiner physischen Entwickelung steht, dafür können als bereite Beispiele dienen folgende ausser Zweifel stehende Daten:

Die Länge des Körpers während der Säuglingsperiode nimmt zu von 50 bis 70 Ctm., oder um 1.4mal, der Umfang der Brust in Höhe der Warzen von 33 bis 45 Ctm., d. h. um 1.36mal, der Umfang des Herzens von 25 bis 45 Cub.-Ctm., also um 1.8mal. Die Körperlänge erreicht während der zweiten Periode von 70 Ctm. an 105 Ctm., in dieser Zeit genau um 1.5mal sich vergrössernd, der Umfang der Brust von 45 bis 56 Ctm., 1.24mal grösser werdend, während der Umfang des Herzens von 45 Cub.-Ctm. bis 78 Cub.-Ctm., d. h. um 1.7mal zunimmt. Die Körperlänge in der dritten Periode steigt von 105 bis 135 Ctm., so sich um fast 1.3mal vergrössernd, der Brustumfang von 36 bis 66 Ctm., d. h. wird um 1.18mal grösser, und das Herz von 78 bis 120 Cub.-Ctm., sich um 1.5mal vergrössernd.

Bei normalen Bedingungen der physischen Entwickelung des kindlichen Organismus kommt auf jedes Kilo Körpergewichtszunahme: in der ersten Periode 3.3 Ctm. Körperwachsthum, 2 Ctm. Brustumfang und 3.3 Cub.-Ctm. Herzumfang; in der zweiten Periode 3.9 Ctm. Körperlänge, 1.2 Ctm. Brustumfang und 3.6 Cub.-Ctm. Herzumfang; in der dritten Periode 1.7 Ctm. Körperlänge, 0.6 Ctm. Brustumfang und 2.3 Cub.-Ctm. Herzumfang.

Auf jeden Ctm. des Umfanges der regelrecht entwickelten Brust kommt an Breite der grossen Herzdämpfung: in der ersten Periode und der zweiten Periode 0.15 Ctm., in der dritten Periode 0.14 Ctm.; linkerseits von der Mittellinie 0.1 Ctm.; rechts 0.05 Ctm. In der dritten Periode entspricht jedem Ctm. Brustumfang 0.14 Ctm. Durchmesser der grossen Dämpfung, wobei auf die linke Seite 0.09 Ctm., auf die rechte 0.045 Ctm. kommen.

Die Länge der untern horizontalen Grenze der grossen Dämpfung, in Bezug auf die genommene Einheit, beträgt 0.12 Ctm. in der ersten und zweiten Periode und 0.11 Ctm. in der dritten.

Die Länge der untern horizontalen Grenze der kleinen Dämpfung beträgt in der ersten Periode 0.6 Ctm., in der zweiten und dritten Periode 0.07.

Auf jeden Ctm. Körperlänge kommt in der ersten und zweiten Periode 0.1 Ctm. Brustbeinlänge, in der dritten Periode 0.09 Ctm. Der obere Theil des Brustbeins, gerechnet vom Einschnitt in Schaft bis zum

Anfang der grossen Dämpfung, beträgt in Bezug auf 1 Ctm. Körperlänge 0.03 Ctm. während aller Kindheitsperioden.

Die Länge der grossen Herzdämpfung bei gegebener Einheit der Körperlänge beträgt am linken Rande des Brustbeins in der ersten und zweiten Periode 0.07 Ctm., in der dritten Periode 0.06 Ctm.

Die Länge der verticalen Kathete der kleinen Dämpfung in der ersten Periode macht 0.04 Ctm. aus, danach 0.05 Ctm.

Von mir ist kein besonderer Unterschied zwischen der Länge der unteren horizontalen Grenze der grossen Dämpfung und der verticalen am linken Sternumrand gehenden gefunden worden.

Wenn auch die soeben von mir gemachten allgemeinen Bemerkungen einigermassen kühn erscheinen können, so verbleibe ich jedoch bei der Hoffnung, ihre Bekräftigung durch Andere zu erleben und diese zwar bei einer grössern Anzahl von Fällen.

Schliesslich halte ich es für meine Pflicht, Herrn Sanatoriumsarzt Dr. I. Kostetzky und Dr. P. Ssukatschew für die mir erwiesene Hülfe bei der Untersuchung der Kinder meinen besten Dank auszusprechen.

L'URICÉMIE CHEZ LES ENFANTS.

PAR LE DR. JULES COMBY (PARIS).

I.

L'INSUFFISANCE des combustions organiques qui se traduit par l'excès d'acide urique et d'urates alcalins dans la circulation a été peu étudiée chez les enfants. C'est dans la goutte de l'adulte surtout que, depuis Garrod, on a poursuivi la recherche de l'acide urique dans le sang, les humeurs, et les tissus de l'économie. Mais la goutte, dans ses manifestations classiques, est exceptionnelle chez les jeunes sujets, et il ne faut pas attendre son apparition pour reconnaître *l'arthritisme* infantile.

Quand, en effet, on étudie de près les descendants d'arthritiques, de goutteux, d'uricémiques, on peut saisir, dès les premières années de la vie, les germes d'une diathèse, d'une dyscrasie latente, qui s'épanouira plus tard avec plus ou moins d'ampleur. Les indices de l'uricémie chez les enfants ne sont pas habituellement d'une netteté absolue, il faut les chercher avec patience et, pour les découvrir, nous devons faire appel à toute notre sagacité de clinicien.

Les difficultés de la tâche expliquent le silence à peu près complet des écrivains médicaux sur l'arthritisme, sur l'uricémie (*lithæmia*) des enfants.

II.

Cependant nous avons le devoir de signaler les tentatives de synthèse et de mise au point faites en France par *H. Cazalis*, aux États-Unis d' Amérique par *Rachford*, etc.

Ce dernier auteur surtout, dans un article très intéressant des *Archives of Pediatrics*, Sept. 1897 ("Symptomatology of Lithæmia"), a reussi à nous donner une description assez complète des états morbides que nous réunissons sous le terme d'uricémie. Avant d'aller plus loin, je résumerai son travail.

Les nouveau-nés éliminent des *urates* en excès dès les premiers jours de la vie, et on peut quelquefois trouver, dans leurs langes, des

sables rouges qui ont été chassés avec les urines. L'urine de ces enfants, très acide, contient des urates et parfois aussi des oxalates en quantité considérable. Des cristaux uratiques se précipitent dans les tubuli des reins, et leur passage à travers les voies urinaires peut les irriter, les blesser, les enflammer.

Le prof. A. Jacobi a fait entrevoir les conséquences proches ou lointaines de ces migrations : albuminurie et néphrite des nouveau-nés, hématuries, calculs de la vessie. Pour prévenir ces accidents, ces complications, et d'autres encore, par exemple la fièvre d'inanition (*inanition fever*—Emmet Holt), la dysurie, l'incontinence ou la rétention d'urine, le spasme de la vessie, l'hydronéphrose, la colique néphrétique, etc., il est nécessaire de faire absorber des liquides aux enfants : lait ou eau. Plus le nouveau-né boira, plus il sera en mesure de balayer son rein, d'en chasser les poussières et graviers qui le souillent. Parmi les accidents uricémiques, M. Rachford distingue surtout des *troubles gastro-intestinaux* et des *troubles nerveux*.

1°. Troubles gastro-intestinaux. — Voici un enfant de souche arthritique (goutte paternelle et maternelle) ; son frère, âgé de 4 ans, a eu de bonne heure des accès soudains, violents mais peu durables, caractérisés par : fièvre, douleurs abdominales, nausées et vomissements, constipation. Au début il y a de l'intolérance stomacale, puis surviennent de la somnolence, de la céphalalgie, de la migraine.

Le petit frère de cet enfant si nettement entaché d'uricémie, n' a que 5 mois et déjà il semble vouloir marcher sur les traces de son aîné. Dès l'âge de 2 mois, il a eu, toutes les 4 à 6 semaines, des troubles digestifs attribués d'abord à la mauvaise qualité du lait, mais se reproduisant en dépit du changement de lait, de la stérilisation de ce lait, de la réglementation des repas. Le 8 Février, il refuse la nourriture ; le 9, vomissements ; le 10, agitation, nausées et vomissements, refus d'alimentation, algidité ; le 11, fièvre, dyspnée, il refuse le lait mais accepte l'eau ; le 12, cris, aggravation ; le 13, calomel à doses fractionnées, un peu de lait est conservé ; le 14, guérison.

L'uricémie peut donc se montrer sous forme de troubles digestifs et on peut avoir des nausées, des vomissements, des selles fétides. Pendant 2, 3, 4, ou 5 jours tout aliment est aussitôt rejeté ; on note de la fièvre, de la prostration, un amaigrissement rapide. L'accès peut se terminer par des convulsions. Les crises sont séparées par des intervalles de santé parfaite durant 1, 2, 3, 6 mois. Toutefois quelques enfants restent pâles, tristes, languissants. Les accès sont variés dans leur forme, mais ils sont souvent marqués par une dyspnée, sans lésion pulmonaire, qui est évidemment d'origine toxique.

2°. Troubles nerveux. — Les enfants uricémiques ont une

certaine précocité intellectuelle, mais ils sont nerveux et irritables. S' ils ont un développement physique satisfaisant, ils n'en sont pas moins exposés à des accidents nerveux dès les premières années. On a relevé plusieurs fois chez eux des attaques éclamptiques. Un enfant de 19 mois a des convulsions suivies de 3 à 5 jours de fièvre, avec vomissements, puis il guérit. De nouveaux accès se déclarent périodiquement, à quelques semaines d'intervalle, et ainsi jusqu' à 4 ans. Alors les convulsions cessent, mais les troubles digestifs augmentent de fréquence et de gravité.

D'après M. Rachford, les accidents nerveux, convulsifs, épileptiformes, seraient provoqués par l'action sur les centres nerveux de la xanthine ou des leucomaïnes ; à côté de l'épilepsie essentielle il y aurait une épilepsie uricémique (*lithæmic epilepsy*), qu'on trouverait associée à la migraine, aux troubles gastriques et aux autres manifestations de la dyscrasie urique.

Parmi les produits toxiques incriminés, M. Rachford distingue l'acide urique et ses composés qui, étant peu ou pas solubles, agiraient localement et les poisons diffusibles (xanthine, para- et hétéroxanthine, etc.). Il a trouvé que, dans la migraine, les paroxysmes coïncidaient avec l'excrétion urinaire de quantités énormes de xanthine. Mais la chimie organique n'a pas dit son dernier mot sur ce point, et les rapports entre les leucomaïnes et les manifestations signalées plus haut ne sauraient être actuellement appréciés avec exactitude.

Je rapprocherai du travail de M. Rachford l'esquisse intéressante que M. le Dr *Whitney* nous a donnée du vomissement périodique (" Cyclic Vomiting ") dans les *Archives of Pediatrics* de Nov. 1898.

Un garçon de 8½ ans, après quelques malaises, un peu de fièvre et d'anorexie, est pris le 23 Juin 1898 de vomissements qui durent 12 heures malgré la diète. Les vomissements étaient muqueux, pituiteux, striés de sang. Amélioration le 3e jour, lait conservé le 4e jour, guérison. Cet accès avait été précédé périodiquement de paroxysmes semblables: 23 Décembre 1896, 20 Mars, 20 Mai, 18 Août, et 18 Novembre 1897. Malgré le régime très sévère, les attaques reviennent tous les 3 mois environ. La première fois (14 Sept. 1896), l'enfant, âgé de 6 ans, fut très malade : pendant 5 jours, vomissements incoercibles avec collapsus, pouls petit et irreguliér, amaigrissement. L'hérédité est très chargée : goutte chez le grand père maternel, tuberculose chez le père, folie chez une tante paternelle, suicide chez un oncle, folie chez une sœur, etc. Enfant nerveux, sans convulsions. Pas de troubles digestifs dans l'intervalle des crises.

Voici la description générale qu'on peut donner de ce syndrome uricémique si bien mis en relief par Mr Whitney. Un enfant,

d'apparence faible ou forte suivant les cas, est pris tout à coup ou après des malaises de courte durée, de vomissements pénibles, incoercibles parfois, d'abord alimentaires, puis muqueux, bilieux, sanguinolents. Toute ingestion d'aliments liquides ou solides est suivie d'un rejet immédiat. Les vomissements se répètent ainsi pendant un jour, 2, 3, 4, 5 jours. En même temps il y a de la fièvre, de l'abattement, un amaigrissement rapide.

Le ventre, loin d'être ballonné comme il l'est dans l'appendicite, la péritonite, l'invagination intestinale, est plutôt affaissé, mou, et rétracté. La crise cesse, l'enfant ne vomit plus, l'appétit revient peu à peu avec la tolérance gastrique, et le retour à la santé est bientôt complet.

Tel est le vomissement cyclique dans sa forme habituelle et typique. Autour des symptômes cardinaux peuvent se grouper des manifestations plus ou moins insolites : céphalalgie, agitation, délire, convulsions, état méningitique, dyspnée, gastralgie, faiblesse et syncope, refroidissement des extrémites, état cholériforme. Dans tous les cas, constipation, d'où le terme de *choléra sec* applicable à certains cas. Ces accès reviennent avec une certaine périodicité, tous les 2, 3, 4, 5, 6 mois, avec santé parfaite dans les périodes intercalaires.

Comment expliquer ces paroxysmes? Il semble bien qu'on soit en présence d'une auto-intoxication uricémique analogue à celle de la goutte, de l'asthme, de la migraine. Les sujets sont d'ailleurs de souche neuro-arthritique. Parfois ils présentent une dyspepsie latente, ils sont gros mangeurs, grands buveurs, exposés à de fréquentes indigestions.

Mr Whitney a donc raison de soigner le régime alimentaire dans l'intervalle des crises, de prescrire la diète absolue quand elles surviennent, en ne revenant que peu à peu l'alimentation normale. Il faut interdire la viande de boucherie, préconiser le régime végétarien, donner des alcalins (salicylate de soude, bicarbonate de potasse), combattre la constipation. Voilà donc, dans la symptomatologie de l'uricémie infantile, deux groupes d'accidents qui se dégagent grâce aux travaux des médecins américains. Je vais maintenant rapporter les faits qui me sont personnels.

III.

J'ai vu des crises périodiques de céphalalgies et de vomissements analogues aux précédents, avec des variantes et des symptômes surajoutés que je signalerai chemin faisant. Un garçon de 12 ans accuse des douleurs de tête qui bientôt le font retirer du collège où il était. Les parents lui font donner des leçons particulières à domicile; le

moindre excès de travail provoque des crises céphaliques, l'anorexie, l'insomnie, la faiblesse générale, et enfin un découragement profond, un état de neurasthénie inquietant. Les nuits sont agitées ; au réveil l'enfant est plus fatigué qu' au moment du coucher. Un séjour prolongé à la campagne, deux villégiatures successives dans les Pyrénées produisent un soulagement momentané. Mais les crises de céphalalgie ont duré plus de 3 ans. Grâce à un bon régime, à l'usage des alcalins, de l'exercice au grand air sans fatigue, la guérison a été obtenue.

Le père de cet enfant est rhumatisant et nerveux ; il a eu des attaques hystériformes. La mère est nerveuse et a eu du diabète sucré. Une sœur plus jeune a des craquements articulaires avec hydarthrose des genoux. L'arthritisme héréditaire et familial est évident.

Dans ce cas les céphalalgies n'ont pas été absolument périodiques, et elles se sont compliquées d'*insomnie*, d'*inaptitude intellectuelle*, et de *neurasthénie* pendant plusieurs années.

Un autre garçon, âgé de 8 ans, très nerveux, très grand pour son âge (taille d'un enfant de 13 à 14 ans), de mère arthritique et eczémateuse, de père goutteux, accuse tout à coup, pendant les vacances scolaires, une *céphalalgie* très vive, mais de courte durée, survenant surtout le soir. Un séjour de trois semaines à la campagne suspend les accidents. Trois mois après, à l'école, l'enfant est repris de mal de tête et souffre aussi du ventre. La crise est soudaine, l'enfant en train de jouer s'arrête tout à coup, porte sa main à la tête et pousse des cris de douleur. Au bout de quelques minutes il peut reprendre ses jeux. La crise se reproduit plusieurs fois par jour sans être suivie jamais de nausées ni de vomissements. Appétit conservé. L'enfant a toujours été un gros mangeur et il affectionne spécialement les viandes de boucherie. On a noté, de temps à autre, des spasmes musculaires, des contractions oculaires, et au début une toux nerveuse, une sorte d'aboiement rappelant la toux hystérique.

Retiré du collège, l'enfant fut traité sans succès par les bromures et l'antipyrine.

L'analyse des urines a montré : densité à + 15°, 1028 ; urée 33 grammes par jour (*azoturie*) ; acide urique 53 centigrammes ; acide phosphorique 3 grammes 20 ; albumine traces ; excès d'urates, etc. Donc il y avait densité et acidité exagérées, azoturie, phosphaturie, *albuminurie légère*.

Un mois après, sous l'influence du régime et des alcalins, les urines se rapprochent de la normale : densité moindre 1024, plus de cristaux d' acide urique dans les sédiments, urée 28 grammes, plus d'albumine. Plus tard, résultat meilleur encore, l'azoturie et l'uricémie disparaissent et la guérison est obtenue.

Voilà donc un cas très net de *céphalalgie paroxystique* qui semble lié à la dyscrasie urique. Elle disparait le jour où les urines redeviennent normales. *L'albuminurie* légère dont il est question plus haut s'est retrouvée dans d'autres observations.

Une fille de 8 ans, grosse et forte, de souche arthritique, ayant eu de l'ictère il y a un an, souffrant d'une constipation opiniâtre, a eu plusieurs accès de *céphalalgie* de courte durée (15 minutes), surtout le soir. Arythmie cardiaque. L'analyse des urines a donné une acidité forte avec 1 gramme 10 *d' acide oxalique* par litre, densité 1024, excès de phosphates et de chlorures, urée 16 grammes 40, traces d' *albumine* (5 centigrammes par litre). Lors d' une précédente analyse faite il y a 8 mois, on avait encore trouvé des traces d' albumine. Guérison par le traitement que j'indiquerai plus loin.

Une fille de 10 ans, grande, élancée, de mère arthritique et dyspeptique, se plaint depuis plusieurs mois de douleurs dans la tête et les membres, qui ont été attribuées à la croissance. Elle mange beaucoup, surtout de la viande. L'analyse des urines donne: *albumine* 22 centigrammes par litre; urée 32 grammes; excès d' acide urique, de phosphates et de chlorures; densité exagérée 1032. Grâce au régime les acidents ont disparu rapidement.

Dans un cas, j'ai trouvé de la *glycosurie* passagère, chez un garçon de 12½ ans, présentant tous les mois ou toutes les six semaines des crises atroces de céphalalgie. L' analyse des urines a donné: densité 1027, glycose 2 grammes, urée 20 grammes, acide urique 67 centigrammes. Il y avait à la fois azoturie, glycosurie, uricémie; tout a disparu sous l'influence du traitement. Les douleurs peuvent occuper, dans quelques cas, les membres, les articulations, et j'ai constaté parfois ce que j' appellerai des *arthralgies uricémiques.*

On me conduit un jour une fille de 14 ans, pesant 60 kilog., ayant un appétit exagére. A 9 ans, cette enfant a eu une métrorrhagie ou des régles précoces; à 10 ans, nouvelle perte, plus rien ensuite. Grands parents obèses. A l' âge de 10 ans, cette enfant a eu, pendant 2 mois, sans fièvre, des douleurs articulaires violentes; elle a souvent du lombago. Les urines sont très acides et contiennent un excès d'urée et d'acide urique. Aux céphalalgies périodiques de l'uricémie, j'ajouterai donc les *arthralgies uricémiques.* Les douleurs vagues dans les lombes et les membres, les ostéalgies, qui, se rencontrant chez des enfants pâles, affaiblis, maigres souvent, parfois obèses, dénotent une intoxication uricémique. Autour de l'acidité exagérée des urines gravitent parfois l'albuminurie, la glycosurie, l'oxalurie, etc.

Chez un enfant uricémique, j'ai observé des *crises hystéro-épileptiques* qui ont guéri. Il est permis de penser que certaines *convulsions*

éclamptiques du premier âge relèvent de l'uricémie. De méme l'excitation cerébrale, les terreurs nocturnes, etc. A côté de ces décharges nerveuses de la dyscrasie acide, il peut y avoir des décharges sur l'appareil urinaire, sur l'intestin, sur le cœur, le poumon, la peau, etc.

Un garçon de 8 ans, nerveux, excitable, ayant des hémorrhoïdes depuis l'âge de 5 ans, de mère obèse, a des spasmes vésicaux, de la pollakiurie, et rend de temps à autre une grande quantité de *sable urique* qui se dépose au fond du vase. Nous avons rappelé plus haut les crises de *vomissements périodiques ;* on peut ajouter certains *flux diarrhéiques* qui, par leur périodicité et leur durée éphémère, font penser à la même cause. De même les *sueurs profuses* survenant soudainement, la *tachycardie* et les palpitations, *l'arythmie cardiaque*, les *crises asthmatiformes* et bronchites sibilantes des enfants arthritiques. Enfin j'ai noté souvent des poussées d' *urticaire*, de *prurigo*, et d'*eczéma* qui relèvent certainement de la même dyscrasie. En voici un exemple. Le 28 Juin 1899, on me conduit un garçon de 9 ans, dont le père est goútteux, graveleux, et obèse (poids 105 kilog.) ; la mère est également obèse, rhumatisante et nerveuse ; un frère âgé de 17 ans, actuellement bien portant, a été diabétique entre 4 et 9 ans ; il aurait eu jusqu' à 60 grammes de sucre par litre.

Notre petit malade, nourri au sein, a eu, depuis l'âge de 6 semaines jusqu' á 4 áns, un eczéma de la face et du corps, qui n'a cédé à aucun traitement. Actuellement il lui reste un état lichénoïde de la racine des cuisses et du scrotum avec recrudescences de temps à autre. Depuis 6 mois, il a des crises de céphalalgie soudaines, peu durables ; il est constipé et a eu des douleurs articulaires. Les urines, très acides, ont une densité de 1034 ; elles sont charéges d' urates.

N'est-il pas légitime de grouper ensemble tous ces accidents, quelle que soit leur localisation, quelle que soit leur forme, quelque dissemblables qu'ils soient en apparence ? N' y a-t-il pas dans les humeurs, dans le sang des malades, une altération identique qui nous révéle le lien pathogénique de tous ces désordres ? Je le crois pour ma part et je pense que l'examen des urines nous autorise à incriminer la combustion incomplète des déchets de l'organisme, c'est-à-dire l'intoxication uricémique (*lithæmia* des Américains). Je vais maintenant, dans une courte synthèse, rassembler les documents épars qui j'ai énumérés, pour aboutir à une thérapeutique rationnelle.

IV.

EXPOSÉ DIDACTIQUE DE L'URICÉMIE INFANTILE.

ETIOLOGIE.—L'hérédité domine la pathogénie de l'uricémie. Quand on étudie l'histoire pathologique des ascendants, on trouve presque

toujours une ou plusieurs affections de la même famille : goutte, obésité, diabète, gravelle, asthme, migraine, névroses, dermatoses, etc.

Toutes les manifestations de la diathèse arthritique se transmettent à la descendance sous une forme identique ou dissemblable (*hérédité homéomorphe* ou *hétéromorphe*). La prédisposition ne s'accuse pas dès la naissance, elle est d'abord cachée, latente, pour se dévoiler plus ou moins tardivement, dans la seconde enfance ou l'adolescence. A la prédisposition innée viennent s'ajouter parfois des influences accidentelles, et l'héritage peut s'enrichir d'acquisitions personnelles. L'hygiène alimentaire doit être mise au premier rang des causes de l'uricémie acquise. L'enfant a mangé trop tôt des aliments trop azotés, trop forts, trop abondants pour son âge. Ses capacités digestives, ses facultés d'assimilation, de combustion organique, ont été dépassées. Il n'a pu utiliser parfaitement ni éliminer suffisamment des aliments ingérés sans mesure ; il a fait de la surcharge graisseuse, il a encombré, irrité les cellules de son foie, de ses reins, de son cerveau, il s'est empoisonné lui-même par les déchets de la suralimentation : vomissements, céphalalgie, albuminurie, convulsions, etc.

L'uricémie est rare dans la première enfance, si l'on en sépare la lithiase rénale, si fréquente chez les enfants mal nourris, athrepsiés. Chez ces bébés, épuisés par la diarrhée, amaigris, déshydratés, les urines se raréfient, se condensent, laissent déposer des urates de soude qui deviennent les noyaux de calculs plus ou moins gros, susceptibles d'entraîner la colique néphrétique, l'hydronéphrose, la dysurie, le calcul urétral, etc. Ici l'influence des troubles digestifs est prédominante ; la tare héréditaire n'apparait pas (consultez le mémoire que j'ai fait paraitre sur cette question dans les *Archives de médecine des Enfants*, Oct. 1899). Ordinairement ce n'est que dans la seconde enfance, comme en font foi les observations citées, que l'uricémie se révéle. Les garçons semblent plus exposés que les filles, mais celles-ci ne sont pas indemnes ; il s'en faut. On remarquera que certaines catégories sociales sont prédisposées, les classes riches, les citadins, les *intellectuels*, les travailleurs de la pensée, qui sont souvent uricémiques par suite de leurs occupations sédentaires, de leur ardeur aux luttes cérébrales, de leurs excès de table parfois. La pathogénie est loin d'être élucidée ; l'acide urique ou lithique (*lithæmia*), élément quaternaire, azoté, est incriminé généralement. Mais on ne sait pas encore bien comment il agit et s'il agit seul dans les phénomènes d'auto-intoxication invoqués par nous. (Consultez sur l'*Uricémie* le livre du Dr Gigot-Suard.—Paris, 1875.)

Symptômes. — L'intoxication uricémique se manifeste par des

symptômes très variables suivant les cas. Ces symptômes peuvent affecter tous les appareils organiques.

1°. Les *symptômes nerveux* ne sont pas les moins fréquents: céphalalgies paroxystiques ou périodiques, douleurs osseuses ou articulaires, excitation cérébrale, insomnie, convulsions, neurasthénie. De tous ces symptômes, le mal de tête est le plus saillant.

2°. Les *troubles digestifs* et les manifestations gastro-intestinales viennent ensuite: vomissements périodiques et incoercibles, coliques, polyphagie, constipation, entérite muco-membraneuse et lithiase intestinale.

3°. Du côté des *voies urinaires* nous avons la lithiase rénale, les coliques néphrétiques, l'albuminurie, la glycosurie, la dysurie et le spasme de la vessie. J'ai vu des enfants, mangeurs de viande, avoir des envies incessantes d' uriner, avec spasmes douloureux, uriner dans leur lit, etc. En somme il y avait de l'irritation vésicale, presque de la cystite du col par concentration des urines; le lait et le régime végetarien faisaient cesser cette manifestation.

4°. *L'appareil respiratoire* participe largement aux manifestations uricémiques: coryza spasmodique, éternuements, pousseès de laryngise et de bronchites sibilantes, accès asthmatiques ou asthmatiformes, congestion pulmonaire, etc.

Le Dr L. H. Watson (*Southern Medical Record*, 1899) regarde l'acide urique comme une cause d' asthme, et il se loue du traitement inspiré par cette théorie.

5°. *L'appareil circulatoire* est quelquefois touché et j'ai observé souvent les palpitations, la tachycardie, l'arythmie cardiaque, la fausse hypertrophie du cœur chez les uricémiques.

6°. *L'appareil tégumentaire* est souvent frappé, car c'est un émonctoire. On note des sueurs abondantes, des éruptions prurigineuses et lichénoïdes, des poussées d' eczéma alternant quelquefois avec des crises d'asthme. L'eczéma tenace et récidivant des enfants est souvent d'origine arthritique et uricémique.

Il y a bien d'autres manifestations possibles; je n' ai énuméré que les principales.

Diagnostic.—Le diagnostic se fait, non seulement par l'étude des symptômes en eux-mêmes, mais encore par la notion des antécédents héréditaires et personnels. En elles-mêmes les manifestations symptomatiques n'ont rien de caractéristique. Ce qui permet de les classer, de les catégoriser, c'est leur filiation, leur parenté, c'est aussi le terrain sur lequel elles ont poussé.

L'anamnèse rendra à ce point de vue les plus grands services, et les renseignements qu' elle fournira seront complétés par l'étude de

l'enfant, de ses attributs physiques, de sa manière d'être, de ses appétits, etc. Enfin l'analyse détaillée des urines viendra éclairer la pathogénie.

PRONOSTIC. — Le pronostic n'est pas grave dans le présent, mais il implique des réserves pour l'avenir. Les uricémiques sont marqués d'une tare constitutionnelle qui appelle toute la sollicitude de l'hygiéniste et du médecin. Leur avenir pourra être bon, médiocre ou mauvais suivant les soins dont ils auront été entourés, suivant la vie qu'ils auront menée. Il dépend d'eux et de leur entourage que la diathèse aille en s'atténuant ou en s'aggravant, qu'elle avance ou retrocéde, qu'elle reste stationnaire ou suive une marche ascendante, qu'elle s'affaiblisse avec les années on au contraire se renforce et engendre des manifestations de plus en plus sévères. Après cela on comprendra l'importance du traitement.

TRAITEMENT. — Avant d'aborder le traitement, disons un mot de la prophylaxie. On est en présence d'un enfant sur lequel pèse un héritage fâcheux. Il n'y a pas lieu d'attendre qu'il ait présenté des accidents pour le soumettre à une hygiène convenable.

L'alimentation sera très surveillée ; après un allaitement naturel prolongé ou un allaitement artificiel bien réglé, on se gardera de commencer trop tôt l'alimentation carnée. Même quand l'enfant est sevré depuis longtemps et témoigne d'une préférence marquée pour les viandes rouges, les mets échauffants, épicés, les sauces de haut goût, on lui refusera ces aliments qui favorisent la production exagérée de l'acide urique.

On écartera surtout la *suralimentation* et on recommandera la plus stricte sobriété. Repas réguliers et en petit nombre (3 ou 4) ; mets choisis et de facile digestion ; pas de vins ni liqueurs alcooliques ; de l'eau ou du lait comme boissons. Régime végétarien mitigé : viandes blanches, poissons, œufs, laitages, légumes farineux, fruits cuits, etc.

On usera très modérément du thé, du café, qui sont énervants. On interdira la charcuterie, le gibier et les viandes faisandées, les salades et crudités, l'oseille, les tomates, etc.

Les fonctions intestinales seront surveillées de très près ; on combattra la constipation par des remèdes si c'est nécessaire, ou l'on cherchera à la prévenir par le régime des légumes verts, des fruits cuits, etc.

On fera fonctionner la peau par les bains, les ablutions froides, le tub, le drap mouillé, les frictions sèches ou alcooliques. On ne négligera pas les exercices physiques ; on prescrira la vie au grand air, le séjour à la campagne ; on évitera la claustration prolongée, la sédentarité, le surmenage cérébral.

Quand la maladie s'est déclarée, s'est manifestée, sous l'une ou

l'autre des formes cliniques étudiées plus haut, on ne manquera pas d'insister sur l'hygiène physique que je viens d'esquisser, et on ajoutera quelques médicaments dont je vais donner la liste.

Les alcalins se recommandent avant tout : bicarbonate de soude ou de potasse, citrate de potasse, magnésie, carbonate ou benzoate de lithine. Ces médicaments seront donnés à doses modérées pendant 10 ou 15 jours de suite, puis repris après un intervalle égal de repos. On se trouvera bien d'ajouter la *noix vomique* (un centigramme de poudre par jour et par année d'âge au maximum). Ce médicament a une action très efficace sur l'atonie du tube digestif, et il s'associe très bien aux alcalins.

Voici une formule dont je me sers volontiers :

Bicarbonate de soude
Magnésie calcinée } āā O gr. 20.
Poudre de noix vomique — *trois centigr.*

Pour un paquet; N° 20. En prendre un matin et soir, avant le repas, dans une cuillerée d'eau sucrée ou de lait (enfant de 6 ans).

Après dix jours, on suspend pour reprendre 10 jours après.

Quand je donne la lithine, j'agis de même; la dose quotidienne de carbonate ou de benzoate de lithine est de 10 centigrammes en moyenne.

Le traitement hydro-minéral joue un rôle important; voici les principales eaux françaises que je conseille suivant les diverses manifestations.

S 'il y a des douleurs, des craquements articulaires, des *arthralgies uricémiques*, on prescrit les eaux chlorurées sodiques chaudes de BOURBONNE (Hte Marne). PLOMBIÈRES (Vosges) convient aux poussées *d'entérite*, aux flux exagérés de l'intestin, aux *douleurs rhumatoïdes abdominales*. CHÂTEL-GUYON (Puy-de-Dôme) se recommande aux enfants constipés, ayant des *selles muco-membraneuses*, du *sable intestinal*, etc. *L'albuminurie* des uricémiques est très efficacement traitée à St. NECTAIRE (Puy-de-Dôme). *La gravelle rénale* est souverainement combattue par CONTREXÉVILLE (Vosges). L'anémie arthritique peut être traitée à ROYAT, La BOURBOULE (Auvergne), St. GERVAIS (Savoie). Les *dermatoses uricémiques* seront envoyées à URIAGE (Dauphiné), LUCHON (Hte. Garonne), La BOURBOULE. Les uricémiques excitables, *neurasthéniques*, relèvent de BAGNÈRES-DE-BIGORRE (Pyrénées).

Les *dyspeptiques* se trouvent bien de VICHY (Allier), POUGUES (Nièvre). Contre certains paroxysmes inquiétants, comme les *vomissements périodiques*, ou agira par la diète absolue, la diète hydrique et si l'estomac vomit tout, on soutiendra l'enfant par des lavements ou des injections sous—cutanées de *sérum artificiel* (eau salée à 7 ou 10 pour

1000). La *céphalalgie paroxystique* imposera le repos absolu de l'enfant, l'interruption des travaux scolaires, sans préjudice des moyens hygiéniques et pharmaceutiques inscrits plus haut.

En résumé, c'est sur l'hygiène qu'il faut le plus compter pour redresser dans la mesure du possible le vice de nutrition qui caractérise *l'uricémie :* hygiène de la digestion, hygiène de la peau, hygiène des muscles, hygiène du corveau. Des aliments bien choisis, en quantité convenable, des repas réguliers, une grande sobriété, des boissons aqueuses ; — des bains, les diverses ressources de l'hydrothérapie, au besoin le massage ; — les jeux de plein air, les exercices physiques ; — repos de l'esprit, pas de surmenage intellectuel.

Avec cela et quelques remèdes bienfaisants pour la digestion et les fonctions intestinales, on obtient beaucoup pour la consolidation d'un organisme mal équilibré et chancelant.

EINIGES ÜBER ESELMILCH ALS SÄUGLINGS-ERNÄHRUNGSMITTEL.

Von H. von RANKE, München.

In der deutschen pädiatrischen Literatur finden sich nur wenige Angaben über Säuglingsernährung mit Eselmilch, so dass nachstehende Mittheilungen einiges Interesse in Anspruch nehmen dürften.

Eine in Paris lebende deutsche Dame, welche seit Jahren an Magenbeschwerden, ohne nachweisbare objektive Grundlage, leidet, deren übrige Organe jedoch gesund sind, gebar am 31. August 1893 ein Mädchen, das bei der Geburt nur 1700 Gramm wog.

Unmittelbar nach der Geburt wurde eine Amme engagirt, eine junge, lebhafte Pariserin, an welcher jedoch das Kind nicht gedieh.

Am 13.	September	betrug	das	Gewicht	1860 g.
" 24.	"	"	"	"	1960 "
" 29.	"	"	"	"	2000 "
" 1.	Oktober	"	"	"	2000 "

Am 1. Oktober wurde die Amme, da sie offenbar nur sehr wenig Milch hatte, entlassen und, auf Anrathen des Arztes, anstatt eines erneuten Versuches mit einer Amme, Eselmilch gegeben, die in Paris stets zu haben ist. Der tägliche Consum des Kindes betrug ca. 1 Liter.

Die bis dahin stets grünlichen und dyspeptischen Ausleerungen wurden nun gelb.

Am 3.	Oktober	war	das	Gewicht	2085 g.
" 8.	"	"	"	"	2325 "
" 14.	"	"	"	"	2680 "

Nachdem so durch die Eselmilch eine entschiedene Wendung zum Besseren eingeleitet war, wurde zu verdünnter, sterilisirter Kuhmilch übergegangen.

Das nun 6 Wochen alte Kind erhielt anfangs 1 Theil Milch zu 2 Theilen dünnem Haferschleim, später halb Milch, halb Haferschleim; erst vom 12. Monat an Vollmilch.

Das Gewicht betrug	am	18.	Oktober	1893 . . .	2810 g.
" " "	"	24.	"	1893 . . .	3005 "
" " "	"	12.	November	1893 . . .	3560 "
" " "	"	10.	Dezember	1893 . . .	4370 "
" " "	"	24.	"	1893 . . .	4660 "
" " "	"	10.	Januar	1894 . . .	5030 "
" " "	"	1.	Juni	1894 . . .	8150 "
" " "	"	12.	Juli	1894 . . .	8250 "
" " "	im Alter von 1 Jahr				9000 "

Das Kind, das, wegen Lebensschwäche, anfangs zu grösster Besorgniss Veranlassung gegeben hatte, erholte sich allmählig in erfreulicher Weise und ist jetzt, November 1899, ein lebhaftes, frisches Mädchen geworden, das in der Entwickelung hinter seinen Altersgenossinnen nicht zurücksteht.

Der Umschwung zum Besseren war durch die Eselmilch erfolgt und die Mutter hatte für diese Ernährungsart eine entschiedene Vorliebe gewonnen.

Als am 27. Mai 1895 das zweite Kind, ein Knabe, geboren wurde, welcher bei der Geburt wieder nur 1740 g. wog, wurde demselben sofort Eselmilch verabreicht; am ersten Tage nur einige Theelöffel voll, später $\frac{1}{2}$ Liter, 1 Liter und zuletzt $1\frac{1}{4}$ Liter täglich.

Am	4.	Juni	betrug das	Gewicht	1610 g.
"	10.	"	"	"	1730 "
"	20.	"	"	"	2020 "
"	27.	"	"	"	2300 "
"	4.	Juli	"	"	2570 "
"	15.	"	"	"	3110 "

Vom 16. Juli an erhielt das etwas über 6 Wochen alte Kind, wie früher seine Schwester, verdünnte Kuhmilch im Soxhletapparat.

Am	11.	September	1895	war das	Gewicht	4450 g.
"	21.	"	1895	" "	"	4860 "
"	16.	November	1895	" "	"	6520 "
"	12.	Dezember	1895	" "	"	6840 "
"	27.	"	1895	" "	"	7050 "
"	27.	Januar	1896	" "	"	7240 "
"	28.	Februar	1896	" "	"	8080 "
"	13.	März	1896	" "	"	8380 "
"	27.	Mai	1896, bei vollendetem ersten Lebensjahre, war das		Gewicht	9250 "

Dieses bei der Geburt gleichfalls lebensschwache Kind hatte also während der ersten 6 Wochen ausschliesslich Eselmilch erhalten und war damit besser gediehen als vorher seine Schwester an der Brust einer indifferenten Amme.

Die Vorliebe der Mutter für Eselmilch hatte dadurch neue Nahrung gewonnen und, als im Sommer 1898 neuerdings eine Niederkunft bevorstand, wünschte die Dame ihr Wochenbett, anstatt in dem heissen

Paris, in Deutschland (Oberbayern), bei ihren Eltern auf dem Lande abzuhalten und das zu erwartende Kind während der ersten zwei Monate mit Eselmilch aufzuziehen.

Zu diesem Behufe wurde aus Südtyrol eine Eselin verschrieben, welche Mitte Juni gefohlt hatte, und mit ihrem Fohlen angekauft.[1]

Am 9. August gebar die Dame ihr drittes Kind, ein Mädchen, das bei der Geburt 2750 g. wog.

Dasselbe erhielt vom ersten Tage an als ausschliessliche Nahrung Eselmilch.

Während der ersten Tage war der Verbrauch etwa ½ Liter, später ca. 1 Liter täglich.

Am 20. August	betrug	das	Gewicht	2820 g.
" 2. September	"	"	"	3084 "
" 11. "	"	"	"	3410 "
" 20. "	"	"	"	3810 "

Die Zunahme hatte also in 40 Tagen 1060 g. betragen; das ergiebt 26.5 g. pro Tag.

Am 21. September erfolgte die Rückreise nach Paris und das Kind erhielt auch dort noch, bis zum 7. Oktober, Eselmilch; ca. 1¼ Liter täglich.

Vom 8. Oktober an wurde sterilisirte Kuhmilch verabreicht, anfangs mit 2 Theilen dünnem Haferschleim vermischt, dann halb und halb, und vom 9. Monat an Vollmilch.

Das Gewicht	betrug	am	15.	Oktober	1898 . . .	4,920 g.
"	"	"	21.	"	1898 . . .	4,890 "
"	"	"	12.	November	1898 . . .	5,140 "
"	"	"	10.	Dezember	1898 . . .	5,990 "
"	"	"	30.	Januar	1899 . . .	7,200 "
"	"	"	9.	August	1899, bei vollendetem ersten Lebensjahre	10,200 "

Sämmtliche 3 Kinder, welche bei der Geburt ein bedeutendes Gewichtsmanco gezeigt hatten, waren also mit Eselmilch, welche während der ersten beiden Lebensmonate verabreicht wurde, in erfreulicher Weise gediehen.

Da mir, nach der Rückkehr der Familie nach Paris, die Eselin zur Verfügung gestellt wurde, wollte ich die Gelegenheit nicht versäumen, auch einige Versuche mit Eselmilchernährung in meiner Klinik bei magendarmkranken Säuglingen anzustellen.

Ich liess daher die Eselin mit ihrem Füllen nach München schaffen

[1] Damit die Eselinnen nicht zu bald ihre Milch verlieren, ist es nöthig, das Fohlen noch an ihnen, wenigstens theilweise, saugen zu lassen. Das Milchquantum, das von einer Eselin zur Säuglingsernährung gewonnen werden kann, ist daher nicht mehr als 1½, höchstens 2 Liter täglich.

und machte in der k. Universitäts-Kinderklinik an 12 durch Gastroenteritis stark herabgekommenen und atrophischen jungen Kindern Versuche mit dieser Ernährung.

Ich lasse diese 12 Fälle, nach ihrem Alter geordnet, mit Angabe des Körpergewichtes, der Diagnose und dem Erfolg der Behandlung hier folgen. Die Gestorbenen wurden sämmtlich obducirt und ist bei ihnen die anatomische Diagnose angegeben.

No.	Alter	Geschlecht	Gewicht	Diagnose	Erfolg der Behandlung
1.	8 Tage	weiblich	2235 g.	Cat. intest.; Icterus, Septicæmie	†
2.	13 "	männlich	1587 "	Atrophische enteritis, Bronchit. capillaris, Bronchopneumonie	†
3.	14 "	"	2246 "	Gastroenteritis	Gebessert entlassen
4.	4 Wochen	"	2832 "	Gastroenteritis	†
5.	4 "	weiblich	1915 "	Chronische Enteritis, Atrophie, Bronchitis	†
6.	5 "	männlich	2454 "	Gastroenteritis, Atrophie	Auf Wunsch der Mutter entlassen
7.	5 "	weiblich	2150 "	Gastroenteritis	Gebessert entlassen
8.	6 "	männlich	2297 "	Gastroenteritis acuta	Geheilt entlassen
9.	2½ Monate	weiblich	3290 "	Gastroenteritis	Geheilt entlassen
10.	5 "	männlich	4500 "	Gastroenteritis	Geheilt entlassen
11.	6 "	"	5470 "	Gastroenteritis, Atrophie, Anaemie, eiterige Bronchitis und beginnende fibrinöse Pleuritis	†
12.	6½ "	"	4300 "	Enteritis follicularis, Anaemie, Atrophie, Gastrectasie	†

Von den 12 mit Eselmilch behandelten, darmkranken und meist stark atrophischen Kindern sind demnach 6 gestorben, während 3 als geheilt, 2 als gebessert und 1 als ungebessert entlassen wurden. Wenn wir letzteres Kind zu den Gestorbenen rechnen, so erhalten wir eine Mortalität von 58.3 %.

Das ist zwar nichts weniger als ein glänzendes Resultat, aber es fragt sich doch, ob man bei diesen an schweren Gastroenteritis leidenden elenden und atrophischen Kindern ein besseres Resultat hätte erzielen können, selbst wenn man im Stande gewesen wäre, ihnen die denkbar beste Nahrung, *i. e.* Frauenmilch, zu verabreichen. Man braucht nur das Alter und das Gewicht der einzelnen Patienten zu vergleichen und bei den Verstorbenen einen Blick auf die anatomische Diagnose zu werfen, um einzusehen, an welch hoffnungslosem Krankenmaterial diese Zahlen gewonnen wurden.

Neben diesen klinischen Erfahrungen über die Wirkung der Eselmilch, war ich im Stande auch über die chemische Zusammensetzung

der Milch der Eselin, welche zu vorstehenden Beobachtungen diente, einigen Aufschluss zu erhalten.

Auf meinen Wunsch hatte Herr Professor Dr. Soxhlet die Güte, die Milch in seinem Laboratorium, durch seinen ersten Assistenten, Herrn Dr. Scheibe, untersuchen zu lassen.

Im Ganzen wurden in der Zeit von Mitte August bis Mitte Dezember 1898 vier Milchproben, sämmtlich Theilmelkungen,[1] chemisch untersucht.

Das Resultat war Folgendes:

In 100 g. Eselmilch waren enthalten:	Probe I.	Probe II.	Probe III.	Probe VI.
Trockensubstanz	10,20	....	....	8,97
Fett	0,68	....	1,17	1,18
Protein	2,32	2,11	1,91	1,31
Milchzucker	6,68	....	....	5,98
Asche	0,59	....	0,47	0,50
Citronensäure	0,105	....	....	....

In 100 g. Eselmilch waren enthalten:	Probe II.	Probe III.
Gesammtstickstoff	0,338 = 2,11 % Protein	0,305 = 1,91 Protein
Eiweissstickstoff (Ritthausen)	.....	0,279 = 1,74 Protein
Nicht-Eiweissstickstoff	.....	0,026.
Caseinstickstoff	0,182 = 1,16 Casein	0,154 = 0,984 Casein
Albuminstickstoff	0,156 = 0,97 Albumin	0,125 = 0,781 % Albumin

An Salzen waren in der Milch enthalten g.:	Probe II.	Probe III.
$P_2 O^5$	1,59	1,38
Chlor	0,21	0,26
Ca O	1,70	1,48
Magnesia	0,16	....
Kali	0,76	0,90
Natron	0,21	....

Spezifisches Gewicht 1,034.

Acidität der frischen Milch 1,8; freiwillige Gerinnung bei Acidität 14,6. Gerinnung beim Kochen bei Acidität 8. Beim Sterilisiren findet Bräunung statt und Ausscheidung eines Niederschlages (Albumin).

Dieser chemische Befund stimmt im Allgemeinen mit dem überein, was man bisher über die Zusammensetzung der Eselmilch wusste, so

[1] Mit dem Ausdruck Theilmelkungen will ich ausdrücken, dass die Milch niemals aus einem Milchgefäss genommen wurde, sondern dass jede einzelne Probe für sich allein in das betreffende Gefäss, dessen Inhalt dann untersucht wurde, gemolken ward.

insbesondere mit den Analysen von Peligot, Vernois et A. Becquerel und Anderen. [1]

Am bekanntesten ist wohl die vergleichende tabellarische Zusammenstellung der Frauenmilch und der Milch verschiedener Thiere, welche freilich noch aus dem Jahre 1852 stammt, die wir aber dennoch hier folgen lassen.[2]

	Spezifisches Gewicht.	Wasser.	Trocken-substanz.	Milch-zucker.	Fett.	Casein und Extractivstoffe.	Salze.
Frauenmilch	1032,67	889,02	110,92	43,64	26,66	39,24	7,38
Kuhmilch	1033,38	864,06	135,94	38,03	36,12	55,15	6,64
Eselmilch	1034,57	890,12	109,88	50,46	18,53	35,65	5,24
Ziegenmilch	1033,53	844,90	155,10	36,91	56,87	55,14	6,18
Stutenmilch.....	1033,74	904,30	95,70	32,76	24,36	33,35	5,23
Schafmilch.....	1040,98	832,32	167,68	39,43	54,51	69,78	7,16

Vernois und Becquerel betonen, dass ihre sämmtlichen Analysen nach ein und derselben Methode gearbeitet seien und desshalb Anspruch auf Exactheit erheben können.

Aus der Tabelle gehe hervor, dass nur in der Eselmilch die einzelnen Substanzen, nach der Menge ihres Vorkommens so geordnet seien wie in der Frauenmilch, nämlich: Zucker, Casein, Fett, Salze, während in der Kuh- und Schafmilch das Casein die erste Stelle einnehme und in der Ziegenmilch das Fett. Man könne daher die Eselmilch als den besten Ersatz für Frauenmilch ansehen.

Diese Anschauung wurde in der That als *mit der Erfahrung übereinstimmend* von den Aerzten acceptirt und festgehalten, doch wurde von den verschiedensten Seiten betont, dass, in Folge ihres geringen Fettgehaltes, die Eselmilch sich als Ersatz für die Frauenmilch *nur für die ersten beiden Lebensmonate eigne.*

Mit dieser Einschränkung steht die Eselmilch, wie wir sehen werden, in verschiedenen europäischen Ländern auch heute noch als Säuglingsernährungsmittel in hohem Ansehen.

Neuerdings kam jedoch Dr. Schlossmann in Dresden auf Grund

[1] M. E. Peligot: "Sur la composition chimique du lait d'ânesse." *Compt. Rend. de l'Academie des Sciences*, Paris, 1836, p. 414.

Vernois et A. Becquerel: "Recherches sur le lait." *Annales d'Hygiène publique et de Médicine légale.* Tome xlix., Paris, 1853, p. 257; Tome l., p. 138.

Simon: *Die Frauenmilch nach ihrem chemischen und physiologischen Verhalten.* Berlin, 1838.

Dumas: *Traité de chimie.* 1843.

Lehmann: *Lehrbuch der physiologischen Chemie*, Bd. i.

Gorup-Besanez: *Eulenburg's Encyklopädie*, ii. Aufl., Bd. v., S. 304.

Duclaux. A. B. Marfau: *Traité de l'Allaitement*, p. 278.

[2] Vernois et Becquerel, a. a. O., T. 50, p. 143.

zahlreicher Analysen, die er im Monat Februar bis 4. März 1897 an der von der "Eselmilchgewinnungs-Genossenschaft Hellerhof" in Dresden verkauften Milch anstellte, zu wesentlich abweichenden Resultaten.[1]

Er fand den Fettgehalt bei weitem niedriger als er bisher angenommen worden war, nämlich nur 0,20 bis höchstens 0,60 %. Auch den Gesammtstickstoff fand er wesentlich niedriger als die bisherigen Untersuchten.

Er weist darauf hin, dass ein Theil der Analysen, die auch heute noch Durchschnittsberechnungen zu Grunde gelegt werden, bereits vor mehr als einem halben Jahrhundert angefertigt worden seien.

Aus der neuesten Zeit, die mit vorgeschrittenen Methoden arbeitet, lässt er, ausser seinen eigenen, nur die Analysen von Munk und Seeliger gelten und kommt zu folgendem Schlusse: Der Hauptunterschied zwischen Frauenmilch und der Milch aller anderen Thiere bestehe in dem Verhältniss zwischen Nhaltiger Substanz und Fett, resp. Nfreier Substanz. Er berechnet dann, auf Grund seiner Analysen, dass ein Säugling im dritten Monat, wenn er die in 920 g. Muttermilch vorhandene Menge Fett mit Eselmilch decken wolle, 10 Liter Eselmilch trinken müsse, oder dass, wenn er 900 g. Eselmilch täglich erhalte, er einen physiologischen Nutzeffect von 294 Calorien, oder fast die Hälfte der, 920 g. Muttermilch entsprechenden, 608 Calorien entbehren müsse.

Schlossmann kommt zu dem Schlusse, dass wir in der Eselmilch durchaus keinen passenden Ersatz der Muttermilch erblicken dürfen.

Dieser Behauptung Schlossmann's muss ich entgegentreten. Nicht als ob ich die Genauigkeit und Zuverlässigkeit der Schlossmann'schen Analysen bezweifeln wollte, aber der Unterschied zwischen seinen Zahlen, besonders bezüglich des Fettes, und denen anderer, ebenfalls nach den neuesten Methoden arbeitender Chemiker ist so gross, dass ich annehmen muss, die von Schlossmann untersuchte Eselmilch habe zufällig ausnahmsweise ungünstige Mischungsverhältnisse geboten. Freilich ist es sehr schwer zu sagen, worin in dem gegebenen Falle diese ungünstigen Verhältnisse begründet gewesen sein können.

Es ist ja bekannt, dass jede Thiermilch, wie auch die Frauenmilch, Schwankungen im Nährstoffgehalte zeigt, je nach der Individualität, dem Zeitpunkt der Lactation, der Art der Futterung, dem Alter des Thieres, der Melkzeit und dem Stadium der Melkung.

In der nachfolgenden Tabelle habe ich die Ergebnisse der neuesten

[1] Arthur Schlossmann: "Ueber Eselmilch." Hoppe-Seyler's *Zeitschrift für physiol. Chemie*, Bd. xxiii., Heft 3, 1897.

Analysen von Soxhlet, Munk,[1] Seeliger,[2] Duclaux,[3] und Schlossmann zusammengestellt.

Aus diesen Zahlen dürfte abzuleiten sein, dass die von Schlossmann auf seine Fettbestimmungen der Eselmilch aufgebaute Berechnung der Calorien auf allgemeine Gültigkeit keineswegs Anspruch erheben kann.

Auch "wenn man gewohnt ist, derartige Fragen wissenschaftlich zu erörtern,"[4] wird man sich hüten müssen, zu seinen Schlussfolgerungen nur das Ergebniss seiner eigenen Analysen zu benutzen,

In 100 Theilen Eselmilch sind enthalten nach

	Soxhlet-Scheibe.				Munk.	Seeliger.		Duclaux.	Schlossmann.
	Probe I.	II.	III.	IV.		I.	II.		
Trockensubstanz	10,20	—	—	8,97	—	—	—	9,30	11,15
Gesammt N	—	0,338	0,305	—	—				0,243
Als Eiweiss fällbare N	—	—	0,279	—	—				0,209
Casein	—	1,16	0,984	—	0,7	1,01	0,654	1,33	0,981
Albumin	—	0,97	0,781	—	1,6		0,37	0,245	0,327
Nicht Eiweiss N	—	—	0,026	—	—	—	—	—	—
Phosphorfleischsäure	—	—	—	—	—		—	—	0,120
Lactoprotein	—	—	—	—	—	0,09	0,07	—	—
Fett	0,68	—	1,17	1,18	1,6	0,45	0,94	1,0	0,364
Zucker	6,68	—	—	5,98	6,0	6,61	4,85	6,5	4,94
Citronensäure	0,105	—	—	—	—	—	—	—	—
Asche	0,59	—	0,47	0,50	0,5	0,42	0,42	0,43	0,309

Auch aus den Seeliger'schen Analysen geht hervor, dass die Eselmilch vom Hellerhof-Dresden besonders fettarm war, doch erhielt S. als Mittel von zwei Analysen einen Fettgehalt von 0,69 %, während bei Schlossmann das Mittel aus allen seinen Analysen nur 0,364 % beträgt. Nach den Analysen aus dem Soxhlet'schen Laboratorium ergiebt sich ein mittlerer Fettgehalt von 1,0 %, bei Munk von 1,6 %, bei Duclaux von 1 %; Gorup-Besanez fand einen Fettgehalt von 1,25, Vernois-Becquerel 1,85, Peligot 1,29.

wenn dieselben mit den Analysen anderer bewährter Forscher so wenig übereinstimmen, und die praktische Erfahrung ganz ausser Acht zu lassen.

Ein englisches Sprüchwort lautet: "The proof of the pudding is in the eating," das dürfte auch hier auf die theoretischen Schlussfolgerungen Schlossmann's anzuwenden sein.

Ich lege grösseres Gewicht auf die praktische Erfahrung als eine, nicht einmal ganz einwandsfreie, Analyse und kann leicht zeigen, dass

[1] Munk und Uffelmann: *Die Ernährung des gesunden und kranken Menschen*, 2. Aufl.

[2] Bei Klemm: "Ueber Eselmilch und Säuglingsernährung." *Jahrbuch für Kinderheilkunde*, xliii. Bd., S. 369.

[3] A. B. Marfan: *Traité de l'allaitement*, Paris, 1899, p. 278.

[4] Schlossmann, *l. c.*

die praktische Erfahrung Anderer mit den von mir mitgetheilten, günstigen Erfahrungen über Eselmilch übereinstimmt.

Dr. Klemm[1] in Dresden hat kürzlich zwei gesunde Kinder, von der Geburt an, ausschliesslich mit Eselmilch ernährt. Das erste hatte bei der Geburt ein Körpergewicht von 3650 g., und erhielt 69 Tage lang ausschliesslich Eselmilch an Stelle der fehlenden Muttermilch. Das Kind consumirte durchschnittlich an einem Tage 865 g. Am Tage der Entwöhnung von Eselmilch betrug das Gewicht 5600 g. Dies ergiebt eine durchschnittliche tägliche Gewichtszunahme von 28 g. Die Uebergewöhnung auf Kuhmilch und Biscuit vollzog sich in der 9. und 10. Lebenswoche ohne Schwierigkeit. Der Knabe ist nicht rhachitisch, hat zur rechten Zeit gezahnt, konnte mit einem Jahre vollständig laufen und wog im Alter von 1 Jahr und 11 Monaten 30 Pfund.

Im Jahre 1898 wurde eine Schwester dieses Kindes gleichfalls vom ersten Lebenstage an, und zwar 64 Tage lang, nur mit Eselmilch ernährt und hat sich ebenfalls gut entwickelt. Auch sie ist nicht rhachitisch und machte bereits im neunten Monat Gehversuche. Zahlen werden für dieses zweite Kind nicht angegeben.

Dr. Klemm schliesst seine Mittheilung mit den Worten: "Die Entwickelung dieses Geschwisterpaares ist für seine Eltern um so bedeutsamer, als sie vorher 2 Knaben im 8. und 9. Monate an Darmkrankheiten verloren hatten. Diese beiden Fälle beweisen, dass die Eselmilch als alleiniges Nahrungsmittel für Säuglinge in den ersten 7 bis 8 Wochen ausreicht."

Klemm hat also an diesen 2 Kindern die gleiche Erfahrung gemacht, wie ich an den 3 oben erwähnten.

Seine Erfahrungen über den Erfolg der Eselmilchanwendung bei magendarmkranken Säuglingen, wie sie in Kinderspitälen aufgenommen zu werden pflegen, decken sich gleichfalls ungefähr mit meinen diessbezüglichen Angaben. Die Gesammtsterblichkeit von 56 magendarmkranken Säuglingen, welche theils im Findelhause, theils in den zwei Dresdener Kinderspitälen mit Eselmilch behandelt wurden, betrug 64,3 %.

Sehr verschieden von dieser hohen Anstaltsmortalität gestaltete sich aber die Sterblichkeit in der Privatpflege. In dieser starben von 123 mit Eselmilch behandelten, verdauungskranken Säuglingen der besser situirten Bevölkerungsklasse nur 13,8 %.

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[1] *Erster Rechenschaftsbericht über die Wirksamkeit des unter dem Schutze Ihrer Majestät der Königin Carola stehenden Hellerhof, gemeinnützige Genossenschaft zur Gewinnung von Eselmilch*, erstattet vom Verwaltungsrathe für die Zeit vom 1. Oktober 1894 bis 31. Dezember 1897. Dresden, 1898, S. 54.

Französische ärztliche Autoritäten äussern sich in demselben Sinne über die Zuträglichkeit der Eselmilch für Säuglinge der ersten Lebensmonate.

So schreibt der bekannte, vorzügliche Pädiater A. B. Marfan in seinem *Traité de l'allaitement et de l'alimentation des enfants du première âge*, Paris, 1899, auf p. 279: "Le lait d'ânesse est celui qui par sa composition se rapproche le plus du lait de femme *et l'observation apprend que les nourrissons le digèrent bien.*"

Er sagt dann weiter: "Le lait d'ânesse est parfois d'une pauvreté extrême, surtout en beurre. Il ne convient plus au nourrisson qui a depassé le troisième mois."

Auf p. 280 äussert er sich folgendermassen: "On est d'abord frappé de ce fait: c'est le lait d'ânesse, qui par la composition quantitative, se rapproche le plus du lait de femme. Ce fait *concorde avec l'observation qui apprend que les nouveaux-nés digèrent mieux le lait d'ânesse que celui d'autres animeaux.*"

In ganz ähnlicher Weise äussern sich eine Anzahl anderer neuerer französischer Autoren über die Bekommlichkeit der Eselmilch für Säuglinge der ersten Lebensmonate,[1] und die Pariser Aerzte im Allgemeinen sprechen sich sehr günstig aus über die praktischen Erfolge, die sie mit Eselmilch erzielen.

Hier ist auf die Thatsache zu verweisen, dass man in Paris täglich Trupps von Eselinnen begegnen kann, welche vor den Thüren der Häuser gemolken werden, damit die Säuglinge die Milch unmittelbar vom Euter weg, frisch erhalten.

Eine der grössten Pariser Zuchtanstalten für Eselmilchgewinnung ist die eines Mr. Letaley, rue Vanneaux 8.

Allerdings ist der Preis dieser Milch ein sehr hoher, durchschnittlich 5 Francs per liter, so dass die Eselmilch nur den wohlhabenden Ständen zugänglich ist.

Allgemein bekannt dürfte es sein, dass Dr. Parrot, der verstorbene, verdienstvolle, erste Professor der Kinderheilkunde an der Pariser Fakultät, im Hôpital des Enfants Assistés, die Einrichtung getroffen hatte, das die syphilitischen Säuglinge direct an das Euter von Eselinnen angelegt und so ernährt wurden. Massgebend war ihm der Gedanke, dass die Eselmilch, nächst der Menschenmilch, die passendste Nahrung für junge Säuglinge sei, während die Syphilis nicht auf das säugende Thier übertragen werden kann, wie etwa auf eine Amme.

Die Erfolge dieser Ernährungsweise wurden anfangs als sehr günstige geschildert. Im ersten Jahre fand eine Abminderung der

[1] Ribblemont, Dessaignes et Lepaye: *Précis d'obstétrique*, Paris, 1896, p. 594.
Tarnier et Chantreuil: *Physiologie et Hygiène de la première enfance*, Paris, 1882, p. 242.

Sterblichkeit von 83,33 %, bei Kuhmilchernährung, auf 30,33 % bei Eselmilch statt.[1]

Trotz dieser Erfolge wurde übrigens nach Parrot's Tode diese Ernährungsweise wieder aufgegeben, wie es scheint, weil die späteren Erfahrungen nicht constant so günstige blieben, hauptsächlich aber wohl wegen der grossen Kosten, welche die Anschaffung säugender Eselinnen veranlasste.

Aehnlich wie in Frankreich hat man auch in Holland günstige Erfahrungen über die Ernährung junger Säuglinge mit Eselmilch gemacht.

In Amsterdam hält ein Herr G. J. Roding, Passeerdwarsstraat No. 14, noch heute einen Eselzuchtstall mit 12 Eselinnen. Früher soll übrigens die Ausdehnung dieses Geschäftes eine sehr viel grössere ge wesen sein.

Auch in Amsterdam, wie in Paris, wird die Milch den Kunden in der Weise geliefert, dass die Eselin vor dem betreffenden Hause gemolken wird. Der Preis ist auch dort ein entsprechend hoher, so dass nur reiche, oder wenigstens wohlhabende Leute von der Eselmilch Gebrauch machen können.

Aus England liegen ebenfalls günstige Erfahrungen vor. So sagt der berühmte Kinderarzt, Charles West, die Eselmilch sei der beste Ersatz für Frauenmilch und, wenn man die Kosten nicht zu scheuen brauche, so sei es rathsam, einem jungen Kinde, das aus irgend welchen Gründen nicht an die Brust gelegt werden könne, wenigstens während der ersten 4 bis 5 Wochen Eselmilch zu verabreichen, bis die ersten Gefahren der künstlichen Ernährung überwunden seien.[2]

Auch die englischen Praktiker machen darauf aufmerksam, dass die Eselmilch, wegen ihres geringen Fettgehaltes, nur für Säuglinge während der ersten 2, höchstens 3 Lebensmonate eine genügende Nahrung darstelle, zur Ernährung älterer Kinder jedoch nicht mehr genüge.

Moore schlägt daher vor, wenn man dennoch ältere Säuglinge mit Eselmilch ernähren wolle, letzterer $\frac{1}{20}$ Rahm zuzusetzen.[3]

Zum Schluss noch ein Urtheil von deutscher Seite aus dem Werke von Munk und Uffelmann: *Die Ernährung des gesunden und kranken Menschen*, Wien und Leipzig, 1887. Diese Autoren geben zwar, als

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[1] E. Thulié: "Rapport sur le service des Enfants Assistés de la Seine." *Progrès médical*, 27, i., 1883.

Lunier et Foville: "L'hospice des Enfants Assistés de Paris." *Annales d'Hygiène publ. et de Médecine legale*, 1891.

[2] Charles West: *Lectures on the Diseases of Infancy and Childhood.* Seventh Edition, 1884, p. 588.

[3] D. Moore: "On the Coagulability of Human Milk." *The Dublin Quarterly Journal of Medical Science*, vol. vii., p. 291.

der menschlichen Milch am nächsten stehend, der Stutenmilch den Vorzug, sagen aber von der Eselinnenmilch, auch sie komme in ihrer Zusammensetzung der Frauenmilch nahe, da sie fast 2% Eiweiss, 1,39% Fett, 6,25% Zucker, und 0,31% Salze führe. Sie fahren fort: "Ueber ihre Verdaulichkeit . . . . wissen wir, dass die Kinder selbst unter ungünstigen Verhältnissen, bei Ernährung mit Eselinnenmilch gut gedeihen."[1]

Also sowohl nach meiner eigenen Erfahrung, als angesichts der Erfahrung vieler anerkannter Praktiker, *kann an der Thatsache kein Zweifel bestehen, dass Eselmilch für Säuglinge der ersten Lebensmonate eine passende und zuträgliche, leicht verdauliche Nahrung darstellt.*

Es fragt sich nun, ob wir aus dieser Thatsache, dass Eselmilch, trotz ihres geringen Fettgehaltes, zur Ernährung in der genannten Altersperiode ausreicht, etwas lernen können.

Zunächst finden wir den Satz bestätigt, den Biedert[2] und Andere schon früher ausgesprochen haben, dass sehr viel kleinere Werthe der Nahrungszufuhr zum Gedeihen junger Säuglinge hinreichen, als man bisher angenommen hatte. Weiter können wir daraus erkennen, dass die wichtigste Bedingung der Zuträglichkeit der Milch für den jungen Säugling nicht sowohl ein hoher Fettgehalt ist, sondern dass das ausschlaggebende Moment in dem Verhältniss der in der Milch enthaltenen Eiweissstoffe zu einander zu suchen ist.

In der Kuhmilch kommen durchschnittlich nach König[3] ("Mittel aus 793 Analysen," S. 295) auf 3,55 % stickstoffhaltiger Substanzen 3,02 % Casein und 0,53 % Albumin; in der Frauenmilch ("Mittel aus 107 Analysen," S. 295) dagegen auf 2,29 % stickstoffhaltigen Substanzen 1,03 % Casein und 1,26 % Albumin; in der Eselmilch ("Mittel aus 19 Analysen," S. 349) kommen auf 2,22 % stickstoffhaltigen Substanzen 0,67 % Casein und 1,55 % Albumin.

Soxhlet (s. Tabelle) hatte (als Durchschnitt von 2 Analysen) in der Eselmilch auf 1,94 % stickstoffhaltigen Substanzen 1,07 % Casein und 0,87 % Albumin gefunden.

Das Casein ist also in der Eselmilch nach König in etwas geringerer Menge vorhanden als in der Frauenmilch, nach Soxhlet etwa in der gleichen Menge; während das Casein in der Kuhmilch, nach Soxhlet, in fast dreimal so grosser Menge, nach König sogar in 4½mal so grosser Menge vorhanden ist als in der Eselmilch.

Auch das Verhältniss des Caseins zum Albumin in der Eselmilch nähert sich vielmehr dem in der Frauenmilch.

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[1] *L. c.*, S. 290.

[2] Biedert: *Die Kinderernährung im Säuglingsalter*, 3. Auflage, 1897.

[3] König: *Zusammensetzung der menschlichen Nahrungs- und Genussmittel*, 1889.

Das Verhältniss des Caseins zum Albumin ist nämlich, unter Zugrundelegung der König'schen Zahlen,

in der Kuhmilch wie 100 : 17,5
in der Frauenmilch wie 100 : 122
in der Eselmilch wie 100 : 231.

Nach den beiden Soxhlet'schen Analysen stellte sich dasselbe Verhältniss wie 100 : 81.

Zu alldem kommt noch, dass bezüglich des Grades der Alkalescenz die Eselmilch der Frauenmilch sehr viel näher steht als die Kuhmilch.

In diesen Factoren liegt offenbar der Schlüssel zur Erklärung der Bekömmlichkeit der Eselmilch für den jungen Säugling, trotz ihres verhältnissmässig geringen Fettgehaltes.

Für die ersten beiden Lebensmonate ist eben das Verhältniss der einzelnen Nahrungsstoffe zu einander, wie sie sich in der Milch finden, wichtiger als die absolute Menge derselben.

Wenn ich es für angezeigt gehalten habe, diese Betrachtungen über Eselmilch zu veröffentlichen, so erübrigt mir zum Schluss noch die Erklärung, dass ich keineswegs erwarte, die Eselmilch nun recht häufig zur Ernährung junger Säuglinge, die aus irgend welchem Grunde die Brust nicht erhalten können, herangezogen zu sehen; einer solchen Einführung stehen wesentliche praktische Hindernisse (hoher Preis und Schwierigkeit der Beschaffung säugender Eselinnen) entgegen. Für die breiten Schichten des Volkes wird daher als Ersatzmittel für Frauenmilch stets nur die Kuhmilch in Betracht kommen, die überall um verhältnissmässig billigen Preis und in beliebiger Menge zu haben ist.

Immerhin dürften, wenn auch nicht häufig, doch zuweilen Verhältnisse sich ergeben, in denen die Kenntniss, dass Eselmilch in der That während der ersten beiden Lebensmonate einen guten Ersatz für die fehlende Muttermilch bietet, eine Richtschnur für unser ärztliches Handeln abgeben kann.

Vor Allem dürften die Betrachtungen, zu welchen wir geführt wurden, bezüglich der Rolle, welche die verschiedenen Eiweissstoffe bei der künstlichen Säuglingsernährung spielen, was ja auch von Biedert und Anderen schon betont wurde, der Beachtung werth sein.

BEMERKUNGEN ÜBER POLIOMYELITIS ANTERIOR ACUTA.

VON PROFESSOR DR. AXEL JOHANNESSEN.

ES giebt einige Krankheiten, die, obschon sie zu den seltener vorkommenden gehören, dennoch vermocht haben, die Aufmerksamkeit auf sich zu lenken, unserer Auffassung bedeutungsvolle physiologischer und pathologischer Prozesse einen weiteren Horizont zu verleihen und den therapeutischen Eingriffen bei einer ganzen Reihe von Krankheiten neue Aufgaben zu stellen.

Zu diesen Krankheiten gehört die Poliomyelitis anterior acuta; und nicht am wenigsten interessant bei diesem Leiden ist der Umstand, dass seine Geschichte in den letzten 60 Jahren eine fortgesetzte Kette von Eroberungen bildet auf Gebieten, die man zu den dunklen der Medicin rechnet.

Zum Verständniss der pathologischen Anatomie der Krankheit ist man auf offenem und geradlinigem Wege vorwärts gegangen. Derselbe begann schon mit v. Heines und Duchennes Annahme, dass der Sitz des Leidens das Rückenmark sei; er wurde durch Cornils, Vulpians, Prévosts, Lockhart Clarkes und Risslers[1] Untersuchungen der vordersten grauen Hörner und deren Ganglienzellen fortgesetzt, und vor allem durch Charcots bedeutungsvolle Arbeiten, sowie die Entwickelung der Anschauungen über diese Krankheit dargestellt ist in der ausgezeichneten Abhandlung von Dr. Mary Putnam Jacobi.[2]

Und das Verdienst des berühmten französischen Forschers bleibt gleich gross, ob auch neuere Untersucher wie Fr. Schultze, Dauber, Goldscheider, Redlich, Simmerling, P. Marie, Harbitz[3] und andere die auch von älteren Verfassern — in erster Linie v. Leyden, Roger und Damaschino — behaupteten Anschauungen bestätigt haben, nämlich,

[1] "Zur Kenntniss der Veränderungen des Nervensystems bei der Poliomyelitis anterior acuta." *Nord. med. Arkiv.* B. xx., No. 22. (1888.)

[2] "Die Pathogenese der infantilen Lähmung." *The Amer. Journal of Obstetrics*, Mai 1874, ref.: *Jahrbuch f. Kinderheilkunde.* B. viii., 1875, S. 203.

[3] Bülow-Hansen und Harbitz: "Beitrag zur Lehre der acuten Poliomyelitis." Ziegler's *Beiträge zur pathol. Anatomie und zur allgemeinen Pathologie.* B. xxv., 1899, S. 517.

dass die Krankheit nicht eine primäre Degeneration der Ganglienzellen in den vordersten Hörnern ist, sondern ein von den Gefässen ausgehender Entzündungsprozess, der wesentlich in den Lokalisationsgebieten der Centralarterien auftritt.

Aber diesen ausgesprochenen Entzündungsprozess mit den stark gefüllten Gefässen, der Rundzelleninfiltration, den kleinen Blutaustretungen und der später erfolgenden Degeneration der Ganglienzellen, wird man in unserer Zeit geneigt sein, als Ausschlag einer Infektion oder Intoxication aufzufassen, und hiermit gelangen wir zu einem der wichtigen Punkte bei der vorliegenden Krankheit, die in der letzten Zeit nicht geringe Aufmerksamkeit auf diese Affektion gelenkt haben.

Wie bekannt, hat bereits Seeligmüller in seiner Monographie vom Jahre 1880[1] ausgesprochen, dass es denkbar sein könnte, dass die Krankheit auf einer toxischen Ursache beruhen könne, indem man dieselbe öfters bei Geschwistern hat auftreten sehen. Im Jahre 1884 wurde diese Anschauung näher entwickelt von Strümpell,[2] der davon ausgeht, dass die multiple Neuritis und Poliomyelitis unter einem einheitlichen ätiologischen Gesichtspunkt aufgefasst werden müssen. Nimmt man nun an, wie es gewöhnlich geschicht, dass die multiple Neuritis infektiöser Natur ist, so hindert kaum etwas die Vermuthung, dass dieselben Krankheitserreger Veränderungen hervorrufen können, die sich bald in den peripheren Nerven, bald im Rückenmark lokalisieren können.

Im Jahre 1889 erhielt diese Auffassung eine bedeutende Stütze durch Risslers obenerwähnte Untersuchungen von vier Todesfällen in Folge akuter Poliomyelitis, wo der Tod 6, 6, 8, und 49 Tage nach Beginn der Krankheit eintrat, und wo Hämorrhagien, parenchymatöse Veränderungen in Leber, Milz und Nieren, Hyperämie und Schwellung der Schleimhaut des Darms gefunden wurde. Ferner wurde bei diesen Fällen[3] dunkles, dickflüssiges Blut, Blutungen unter der Pleura und dem Endocardium und Myocarditis nachgewiesen — alles zusammen Zeichen, die auf eine allgemeine Infektion hindeuteten.

Es dauerte denn auch nicht lange, ehe man mit bakteriologischen Untersuchungen frischer Fälle begann und mit Versuchen, analoge Zustände bei Tieren durch Inokulation oder intravenöse Injektionen mit Bakterien oder Toxinen hervorzurufen. Es zeigt sich nun, dass es durch solche Experimente gelingt, Rückenmarkskrankheiten

[1] "Spinale Kinderlähmung." Gerhardt: *Handbuch der Kinderkrankheiten.* B. v., 1. Abtheilung, 2. Hälfte, S. 105.

[2] "Ueber das Verhältniss der multiplen Neuritis zur Poliomyelitis." *Neurologisches Centralblatt*, 1884, S. 241.

[3] Vergl. Medin: "Om den infantila paralysien med särskild hänsyn til dess acuta stadium." *Nord. med. Arkiv*, 1896, S. 72.

hervorzurufen, die denen des Menschen gleichen, so z. B. mit Bacterium coli commune (Gilbert und Lion), Streptokokken (Roger), Erysipelaskokken (Bourges), Bacterium coli commune und Staphylococcus pyogenes aureus (Thoinot und Masselin). Aber die injicierten Bakterien wurden in den Körpern der Tiere nicht wiedergefunden.

Bei den vorliegenden bakteriologischen Untersuchungen bei Menschen wurden weder in Deckglaspräparaten, in Schnittpräparaten noch in Kulturen aus der Rückenmarksubstanz auf Serumagar, Agar und Bouillon irgend welche Bakterien gefunden. (Simmerling, Goldscheider, Harbitz).

Der letztgenannte dieser Untersucher[1] fand indessen in einer Kultur aus Cerebrospinalflüssigkeit, die zu Lebzeiten des Patienten herausgenommen war, einen eigenthümlich aussehenden Diplococcus oder ein kurzes Doppelstäbchen, welches nach Gram gefärbt wurde.

Auch Schultze[2] hat in Cerebrospinalflüssigkeit von einem lebenden Individuum Bakterien gefunden, aber in diesem Falle war es im Gegensatz zum erstgenannten Funde leicht, dieselben als Weichselbaum-Jägersche Meningokokken zu bestimmen. Ob man hierauf weitere Schlussfolgerungen bauen darf, muss die Zeit zeigen.

Aber vorläufig kann man noch nicht auf irgend eine geschlossene Beweiskette für die bakterielle oder toxische Natur der Krankheit hinweisen; wenn trotzdem die Auffassung derselben als eine Infektionskrankheit immer mehr Ausbreitung gewinnt, so ist dies in nicht geringem Grade gewissen Eigenthümlichkeiten bei ihrem Auftreten und Vorkommen zu verdanken.

Es zeigt sich nämlich, dass mehrere Fälle fast zur selben Zeit beobachtet werden können, so dass man an eine Art epidemischen Vorkommens denken muss; und demnächst sind es wiederum gewisse Zeiten des Jahres, wo das Vorkommen häufiger als zu anderen Zeiten ist.

Wesentlich scheinen die Sommer- und Herbstmonate die Zeit zu sein, da man die meisten Fälle entstehen sieht.

Die ersten Beobachtungen dieser Verhältnisse verdankt man, wie es scheint, amerikanischen Aerzten. So z. B. bemerkt nämlich Wharton Sinkler,[3] dass die spinale Kinderlähmung in Philadelphia vornehmlich in der heissen Jahreszeit (47-mal unter 57 Fällen) vorkommt und Colmer[4] berichtet über einen ärztlichen Besuch, den er im

[1] a. a. O., S. 543.

[2] "Zur Aetiologie der akuten Poliomyelitis." *Muench. med. Wochenschrift*, 1898, No. 32.

[3] "Palsies of children." *Amer. Journal of the Med. Sciences*, April, 1875. Citiert nach Seeligmüller, a. a. O., S. 100, und *Jahrbuch für Kinderheilkunde*, B. ix., 1876, S. 191.

[4] *Amer. Journal of the Med. Sciences*, 1843, B. v., S. 248. Citiert nach Medin, a. a. O., S. 42.

Jahre 1841 in "the parish of West Feliciana, La., U. S.," vornahm, wobei er einen einzelnen Fall von Lähmung bei einem Kinde von 1 Jahr sah. Bei dieser Gelegenheit erzählten die Eltern von 8—10 anderen Fällen derselben Krankheit, die im Laufe von 3—4 Monaten bei Kindern unter 2 Jahren im Umkreise weniger Meilen vorgekommen sein sollten.

Der erste europäische Arzt, der eine Epidemie von Poliomyelitis beschrieben hat, ist ein schwedischer Bezirksarzt, Dr. Bergenholtz in Umeå, im nördlichen Theile Schwedens. In seinem Medicinalbericht für's Jahr 1881 an die schwedische Medicinalverwaltung führt er an, dass er 18 Fälle dieser Krankheit beobachtet hat. Von diesen Fällen kamen 3 im Juli, 10 im August, 4 im October und einer im November vor.

Die nächste Mittheilung stammt aus Frankreich, indem Cordier[1] eine kleine Epidemie beschreibt, die im Jahre 1885 in einem Städtchen von 14—1500 Einwohnern, Namens Sainte-Foy-l'Argentière in der Nähe von Lyon beobachtet wurde.

Im Laufe der Monate Juni und Juli traten hier wenigstens 13 Fälle auf, im Alter von einem Monat bis 2½ Jahre. Von diesen starben vier. Die Krankheit wurde wahrscheinlich durch persönliche Ansteckung übertragen. In einzelnen Fällen gelang es die Dauer der Inkubationszeit zu bestimmen, die in einem Fall 36 Stunden betrug; 2 Kinder, die das angegriffene Städtchen besucht hatten und am Abend nach Hause reisten, wurden in derselben Nacht krank.

Gleichzeitig hat auch Strümpell[2] 3 Fälle, wovon 2 Geschwister, im Laufe von 10 Tagen bei Kindern in einem kleinen Dorfe beobachtet.

In Norwegen trat im Juli bis September 1886 eine kleine Epidemie auf in der Stadt Mandel und Umgegend, im südlichsten Theil des Landes. Der behandelnde Arzt, Dr. Oxholm,[3] beobachtete selbst 5 Fälle, die in einem Alter von ½—4 Jahren waren und hörte von 4 gleichzeitig vorkommenden.

Ferner berichtet Leegaard,[4] dass in Eidsvold (Akershus Amt) ein 8jähriger Knabe und 4 Tage später ein 20jähriger Arbeiter auf demselben Hofe erkrankte und, dass am 24. October, 1889, in Ski ein

[1] "Relation d'une épidémie de paralysie atrophique de l'enfance." *Lyon méd.*, B. lvii., 1888, 1–2. Citiert nach Schmidt's *Jahrbücher*, Jahrgang 1888, B. ccxviii., S. 29.

[2] *Festschrift* für E. Wagner, S. 217. Citiert nach Schmidt's *Jahrbücher*. B. ccxviii., 1888, S. 30.

[3] *Beretning om Sundhedstilstanden og Medicinalforholdene i Norge 1886*, S. 102, og Oxholm: "Tilfälde af omtrent samtidig opträdende Lammelser hos Börn." *Tidsskrift for praktisk Medicin*, 1887, S. 193.

[4] "Om Poliomyelit med Demonstration af mikroskopiske Preparaster." *Forhandlinger og Foredrag paa det 3de norske Lägemöde 1889*, S. 80.

9jähriger Knabe und kurze Zeit darauf ein 37jähriger Kätner erkrankte, gleichwie auch ähnliche Fälle in Lärdal, Sjövik, und Rakkestad vorgekommen sein sollen.

Im Jahre 1887 traf eine bedeutende Epidemie in Stockholm [1] ein. Von den vorkommenden Fällen sind 43 bekannt; von diesen trafen 2 im Mai, 1 im Juni, 2 im Juli, 15 im August, 16 im September, 5 im October, 2 im November ein. Die meisten Fälle, nämlich 30, kamen vom 9. August bis 30. September vor. 4 starben.

Im	Jahre	1888	trafen	in	Stockholm	7	Fälle	von	Kinderlähmung ein.
"	"	1889	"	"		11	"	"	" "
"	"	1890	"	"		6	"	"	" "
"	"	1891	"	"		9	"	"	" "
"	"	1892	"	"		5	"	"	" "
"	"	1893	"	"		8	"	"	" "
"	"	1894	"	"		3	"	"	" "

Im Jahre 1895 dagegen 21 Fälle, wovon 2 im März, 1 im Juni, 4 im Juli, 4 im August, 8 im September, 2 im October.

Es scheint also auch in diesem Jahre ein epidemisches Auftreten der Krankheit stattgefunden zu haben.

Von den während der beiden Epidemien angegriffenen 64 Patienten waren:

20 unter 1 Jahr.	1 von 4—5 Jahren.
17 von 1—2 Jahren.	3 " 5—6 "
13 " 2—3 "	1 " 7—8 "
9 " 3—4 "	

Von den Angegriffenen waren 37 Knaben und 27 Mädchen.

Im Jahre 1889 trafen in Jena und Umgegend 5 Fälle von Poliomyelitis ein, wovon einer im Juni und die übrigen 4 im Juli auftraten. Die Kinder waren in einem Alter von 9 Monaten bis 3 Jahren.[2]

Im Jahre 1894 beschreibt Macphail [3] eine eigenthümliche Krankheit, die im Staate Vermont und in Canada epidemisch auftrat, und die im Laufe vom Juni bis August 120 Personen angriff, wovon 6 Erwachsene und die übrigen Kinder. Der Todesprozentsatz war 13%. Anfangs nahm man an, dass die Krankheit epidemische Cerebrospinalmeningitis sei, später nahm man wegen des ganzen Verlaufs der Krankheit, der Art und Verbreitung der Lähmungen, sowie der elektrischen Verhältnisse an, dass es sich um eine Epidemie von spinaler Kinderlähmung handelte.

[1] Medin. a. a. O.

[2] Briegleb: "Ueber die Frage der infektiösen Natur der akuten Poliomyelitis." *Inaug. Dissert.* Jena, 1890.

[3] "A Preliminary Note on the Epidemic of Paralysis in Children." *British Medical Journal*, 1894, vol. ii., S. 1233.

Die Fälle sind jedoch nicht so genau beschrieben, dass man den bestimmten Schluss ziehen könnte, dass wirklich eine Poliomyelitis vorgelegen hat; bemerkenswerth ist auch, dass 12 Pferde an "Cerebrospinalmeningitis" starben, und dass einige wenige Fälle von "Paralysis" beim Geflügel beobachtet wurden.

In Italien hat Pierracini[1] in einer kleinen Stadt in der Nähe von Florenz im Laufe von 15 Tagen 7 Kinder an spinaler Lähmung erkranken sehen und Bucelli[2] in Genua im Laufe von 4 Monaten 17. Er führt auch an, dass es sich beim Zusammenstellen von 129 Fällen, die in den städtischen Krankenhäusern behandelt wurden, ergab, dass die meisten Fälle im August vorkamen.

Pleuss[3] hat in Kiel und Umgegend 4 Fälle von Juni bis October 1897 bei Kindern von 4 Monaten bis 4 Jahren beobachtet, und Pasteur[4] hat gesehen, dass in einer Familie 7 Kinder von einer eigenthümlichen Affektion angegriffen wurden, die bei 2 als eine Poliomyelitis, bei 1 als eine Cerebral-Lähmung, bei 2 als ein Strabismus internus mit Tremor und bei 2 als eine akute febrile Krankheit auftrat.

Schliesslich berichtet Auerbach[5] über ein Vorkommen von 9 Fällen in Frankfurt a. M. in den Monaten Mai bis December 1898; von diesen traten 4 in derselben Strasse auf. Ausserdem waren 3 Fälle, die im Jahre 1897 und 2, die im März 1898 begonnen hatten, beobachtet worden. Die Angegriffenen waren im Alter von 8 Monaten bis $2\frac{1}{2}$ Jahren. Die Anzahl der Knaben betrug 9.

Aus diesen verhältnismässig wenigen Berichten wird hervorgehen, dass ein Vorkommen der Krankheit mit fast epidemischem Charakter nicht so häufig zu sein scheint, als dass man mit Grund erwarten kann im praktischen Leben darauf zu stossen. Man wird darauf angewiesen sein, die Krankheit während ihres allgemeinen Auftretens zu studieren, so wie dieselbe sich in Krankenhäusern und Polikliniken zeigt, und das Interesse wird sich hier darum sammeln, inwiefern man auch ausserhalb der "Epidemien" die eigenthümlichen Verhältnisse, die ihrem Auftreten während derselben zu folgen scheinen, gewahr werden kann.

Von dieser Auffassung ausgehend, werde ich mir gestatten, die

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[1] Lo speriment 1895, citiert nach Pleuss: "Ueber gehäuftes Vorkommen spinaler Kinderlähmung." *Inaug. Dissert.*, Kiel, 1898, S. 13.

[2] Policlinico 1897, No. 12, ref. in *Deutsche med. Wochenschrift*, 1898, Lit. Beilage 11.

[3] a. a. O.

[4] "Cases of Infantile Paralysis." *Clinical Transactions*, vol. xiii. Citiert nach Virchow *Jahresbericht über die Leistungen und Fortschritte in der gesammten Medicin*, 1897, B. ii., 2. Abth., S. 664.

[5] "Ueber gehäuftes Auftreten und über die Aetiologie der Poliomyelitis anterior acuta infantum." *Jahrbuch für Kinderheilkunde*, B. l., 1899, S. 41.

Fälle, welche auf der pädastrischen Universitätsklinik in Christiania von 1893–1899 vorgekommen sind, zu besprechen.

Es sind in diesem Zeitraume 23 Fälle auf der stationären Klinik vorgekommen. Von diesen waren 16 Knaben und 7 Mädchen.

Das Alter der Knaben bei der Aufnahme in die Klinik war folgendes (vergl. Tabelle, Seite 40):

1 Jahr:	1	4–5 Jahre:	6
1–2 Jahre:	3	5–6 "	0
2–3 "	2	6–7 "	2
3–4 "	1	7–8 "	1

Das Alter der Mädchen bei der Aufnahme in die Klinik:

1–2 Jahre:	1	9–10 Jahre:	1
4–5 "	1	11–12 "	2
7–8 "	2		

Die Krankheit entstand dagegen bei den Knaben in einem Alter von

4 Monaten bei	1	3–4 Jahren bei	2
1–2 Jahren "	9	4–5 " "	1
2–3 " "	3		

Bei den Mädchen in einem Alter von

4 Monaten bei	1	3– 4 Jahren bei	1
8 " "	1	9–10 " "	1
1–2 Jahren "	2	Unbekannt "	1

Der Abstand zwischen der Zeit, da der Angriff stattfand und dem Eintritt ins Krankenhaus war in Betreff der Knaben folgender:

In 1 Fall	1 Monat	In 1 Fall	$2\frac{10}{12}$ Jahre
" 1 "	2 Monate	" 2 Fällen	3 "
" 2 Fällen	3 "	" 1 Fall	4 "
" 4 "	6 "	" 1 "	$4\frac{1}{2}$ "
" 2 "	1 Jahr	" 1 "	6 "

In Betreff der Mädchen:

In 2 Fällen	$0-\frac{1}{4}$ Jahr	In 2 Fällen	10 Jahre
" 2 "	$6\frac{1}{2}$ Jahre	" 1 Fall	unbekannt

Fast die Hälfte der Fälle, nämlich 11, trafen im Alter von 1–2 Jahren ein. Die übrigen Fälle sind zwischen die übrigen Altersklassen bis zu 4 à 5 Jahren verteilt. Nur ein einzelner Fall zeigt ein höheres Alter, nämlich 9 Jahre. Das jüngste Alter, worin die Krankheit beobachtet wurde, war 4 Monate, zu welcher Zeit dieselbe bei 2 Individuen—einem Knaben und einem Mädchen—auftrat. Dies stimmt gut mit früheren Erfahrungen. So z. B. erwähnt Seeligmüller,[1] dass die Krankheit am allerhäufigsten im ersten und zweiten Jahre auftritt und Goldscheider,[2] dass Kinder von 1–2 Jahren am häufigsten angegriffen werden. Medin [3]

[1] a. a. O., S. 100. [2] a. a. O., S. 429. [3] a. a. O., S. 48.

hat die meisten Fälle, nämlich 20, in der Altersgruppe 0–1 Jahr beobachtet, während er blos 17 von 1–2 Jahren sah.

Das Verhältniss zwischen angegriffenen Knaben und Mädchen war wie 2,3 : 1, was ziemlich genau den Zahlen entspricht, welche Medin bei der Epidemie von 1895 beobachtete, wo 15 Knaben und 6 Mädchen erkrankten.

Während der Epidemie von 1887 dagegen war die Anzahl beider Geschlechter ungefähr gleich, indem 22 Knaben und 21 Mädchen angegriffen wurden.

Bei Seeligmüller's Patienten war das Verhältniss zwischen Knaben und Mädchen wie 4 : 3.

Untersucht man die 23 Fälle nach der Zeit und dem Orte, wo die Krankheit begonnen hat, wird man folgende Reihen erhalten:

Im Jahre	1887, Juli,	wurden	angegriffen:	1 in Christiania.
"	1888, "	"	"	1 " "
"	1888, August,	"	"	1 " "
"	1889, "	"	"	1 " "
"	1889, Sommer,	"	"	1 " "
"	1890, "	"	"	1 " Dröbak.
"	1891, "	"	"	1 " Drammen.
"	1892, Herbst,	"	"	2 " Christiania.
"	1894, "	"	"	1 während eines Aufenthaltes in Georgia. in Nord-Amerika.
"	1894, Mai,	"	"	1 in Bärum (Akershus Amt).
"	1895, September,	"	"	1 " Eidsvold (Akershus Amt).
"	1896, Mai,	"	"	1 " Christiania.
"	1896, Juli,	"	"	1 " "
"	1896, August,	"	"	1 " "
"	1896, "	"	"	1 " Näs, Romerike (Akershus Amt).
"	1896, "	"	"	1 " Trögstad, (Smaalenenes Amt).
"	1896, "	"	"	1 während eines Aufenthalts in Eidsvold.
"	1896, September,	"	"	1 in Enebak (Akershus Amt).
"	1897, August,	"	"	1 " Sörum " "
"	1897, September,	"	"	1 " Christiania.
"	1897, December,	"	"	1 " "
Unbekannt				1 " Skien.

Aus dieser Zusammenstellung wird hervorgehen, dass im Jahre 1896 offenbar eine grössere Verbreitung der Krankheit in Christiania und den angrenzenden Bezirken stattgefunden hat, indem nicht weniger als 7 Individuen, die in diesem Jahre angegriffen waren, in der Klinik behandelt wurden. Gleichfalls scheint auch das Jahr 1897 mehr Fälle dargeboten zu haben, als gewöhnlich in den anderen Jahren beobachtet wurden. Nach dem, was ich in Erfahrung gebracht habe, ist auch von anderer Seite eine grössere Häufigkeit der Krankheit in diesen Jahren, als sonst in den genannten Ortschaften bemerkt worden, so dass man

möglicherweise annehmen darf, dass dieselbe wirklich eine Art epidemischen Charakters gehabt hat.

Was Jahreszeiten und Monate betrifft, ist die Krankheit folgendermassen aufgetreten:

In den Frühjahrsmonaten	März–Mai	in	2 Fällen.
" Sommermonaten	Juni–August	"	10 "
" Herbstmonaten	September–November	"	5 "
" Wintermonaten	December–Februar	"	1 Fall.
Unbekanntes Entstehen		"	5 Fällen.

Der Monat, wo die Krankheit am häufigsten begonnen hat, ist der August. Wie man bemerken wird, stimmt dies sehr gut überein mit den oben referierten Beobachtungen in früheren Epidemien.

Es sieht also so aus, als ob es eine ziemlich allgemeine Erscheinung sei, dass die Individuen, die am häufigsten angegriffen werden, ganz kleine Kinder von ungefähr 1 Jahr sind, und dass sie vornehmlich in den warmen Monaten des Jahres—wesentlich Juli und August—angegriffen werden.

Aber die Krankheiten, welche häufig Kinder im genannten Alter in den Sommer- und Herbstmonaten angreifen, sind Affektionen im Verdauungskanal, und es liegt daher nahe zu fragen, inwiefern ein Zusammenhang zwischen den gastro-intestinalen Leiden und der Poliomyelitis denkbar sei.

Nun ist es freilich der Fall, dass im initialen Fieberstadium häufig gastrische Symptome beobachtet werden, gleichwie oft angeführt wird, dass der Patient in diesem Stadium[1] an Diarrhoe oder Stuhlverstopfung gelitten hat. Aber die Frage ist für uns gewesen, ob eine tiefere Verbindung zwischen diesen Leiden nachgewiesen werden kann,—mit anderen Worten ob dieselbe Ursache beide Krankheiten hervorbringen kann, oder ob die gastro-intestinale Affektion den Pass bildet, durch welchen die Poliomyelitis in den Körper dringt.[2]

In der oben citierten Arbeit von Bülow-Hansen und Harbitz werden 2 Fälle—Geschwister—besprochen, die am 7ten, resp. 5ten Tage nach Beginn der Krankheit starben, und wo Symptome aus dem Verdauungskanal vorhanden gewesen. Es wurde bei diesen folgendes gefunden: In dem einen Falle geschwollene Mesenterialdrüsen, geschwollene und hyperämische Peyersche Plaques im Dünndarm; im anderen Falle etwas Hyperämie und Schwellung der Peyer'schen Plaques, während das lymphatische Gewebe im Ileum stark hyperämisch und geschwollen war, gleichwie auch mikroskopisch starke Hyperämie und Rundzelleninfiltration im Dünndarm nachgewiesen wurde.

[1] Vergl. Seeligmüller, a. a. O., S. 47, Goldscheider, a. a. O., S. 413, Medin, a. a. O., S. 51.

[2] Vergl. Johannessen: "Om Poliomyelitis, Foredrag i det med. Selskob i Christiania, Mai 1898." *Forhandlinger i det med. Selskab*, 1898, S. 81.

Ähnliche Funde sind auch bei zweien von Medins Patienten, einem $4\frac{1}{2}$ Jahre alten und einem 5 Monate alten Mädchen, nachgewiesen, die beide am 6ten Tage an der Krankheit starben.

Die erwähnten norwegischen Verfasser nehmen mit Rücksicht hierauf an, dass der krankhaft afficierte Darmkanal wirklich die Eingangspforte ist oder wenigstens sein kann.

Es wird jedoch einleuchtend sein, dass es angesichts der geringen Anzahl von Sektionen der Todesfälle[1] im akuten Stadium kaum möglich sein wird, auf diesem Wege zu einem bestimmten Resultat zu gelangen. Man muss sich bis auf Weiteres mit der klinischen Beobachtung begnügen. Nun ist es freilich der Fall, dass einzelne Beobachtungen von Interesse vorliegen, z. B. in einem von Briegleb[2] angeführten Falle, wo ein 3 Jahre altes Mädchen Cholera nostras gleichzeitig mit 3 anderen Mitgliedern der Familie bekam. Bei diesen letzteren verlief die Krankheit normal, während bei dem kleinen Mädchen 4 Tage später das rechte Bein gelähmt wurde. Aber solche Fälle kommen nicht häufig vor.

Bei Untersuchung unserer Fälle zeigte es sich auch, dass bloss bei einem einzelnen Patienten—einem 4 Monate alten Knaben—starke Diarrhoen vor Ausbruch der Krankheit beobachtet wurden, gleichwie die Darmentleerungen auch nach Auftreten der Lähmungen blutvermischt waren.

Bei einem $3\frac{1}{2}$jährigen Knaben trat 8 Tage vor Beginn der Krankheit eine hartnäckige Obstipation ein, verbunden mit Schmerzen im Unterleibe und Schwierigkeit beim Harnlassen. Aber abgesehen von den initialen Erbrechungen wurden in keinem anderen Falle intestinale Störungen beobachtet.

Dagegen stellte die Krankheit sich bei einem 1 Jahre alten Mädchen in unmittelbarem Anschluss an eine Pneumonie ein, was auch früher beobachtet worden, unter anderen von Pleuss[3] bei einem 4 Monate alten Knaben.

Bei einem $3\frac{1}{2}$jährigen Mädchen fand sich die Krankheit ein sich nach einer eigentümlichen Affektion von Labia majora, die geschwollen, blauschwarz und schmerzhaft waren.

In den übrigen Fällen wird angegeben, dass die Kinder keinerlei Krankhaftes dargeboten hatten, ehe die Krankheit über sie kam. Man muss doch hier wohl bedenken, dass in mehreren Fällen längere Zeit verstrichen war, ehe sie in die Klinik gebracht wurden, so dass die Angaben wohl nicht immer völlig zuverlässig sind.

[1] Ausser den genannten sind referiert: 1 von Drummond, 1 von Goldscheider, 1 von Dauber, 1 von Redlich, 2 von Siemerling, 1 (erwachsenes Mädchen) von Medin.

[2] a. a. O., S. 17.

[3] a. a. O., S. 15.

Aber da die Aufmerksamkeit hinsichtlich dieser so interessanten Fragen über die Eingangspforten der Krankheit geweckt worden sind, steht es wohl zu erwarten, dass die Untersuchungen hierüber mit grosser Genauigkeit sowohl klinisch als auch pathologisch-anatomisch ausgeführt werden.

Medin bemerkt,[1] dass die meisten seiner Fälle der Arbeiterklasse angehörten, jedoch keineswegs den eigentlich armen Familien. Dieselbe Erfahrung haben auch wir gemacht, indem wir bloss in einem einzelnen Falle die Krankheit bei einem Kinde in armen und verkommenen Verhältnissen auftreten sahen. In Betreff der übrigen Fälle war die Verteilung nach der Stellung des Vaters in der Gesellschaft folgende:

In 5 Fällen Arbeiter.
" 1 Fall Hafenarbeiter.
" 3 Fällen Tischler.
" 1 Fall Zimmermann.
" 1 " Maler.
" 1 " Maurer.
" 1 " Schuhmacher.
" 1 " Bäcker.
In 1 Fall Typograph.
" 1 " Fuhrmann.
" 1 " Brodführer.
" 1 " Müller.
" 1 " Landmann.
" 1 " Zollaufseher.
" 1 " Unehelich, aber in einer Arbeiterfamilie aufgenommen.

Was die Verwandtschaft im Übrigen betrifft, so war in 2 Fällen ausgesprochene Tuberkulose in der Familie beobachtet; in 1 Falle war der Vater an dieser Krankheit gestorben.

In 1 Falle war der Vater und in 1 Falle die Mutter neurasthemisch. In 1 Falle litt die Mutter an Hemikranie und der Vater an Arthritis.

In 1 Falle waren 2 Schwestern des Vaters irrsinnig.

In 1 Falle litt die Mutter und eine Cousine derselben an Epilepsie.

In 1 Falle litt ein Bruder des Vaters an Lähmungen nach einer Poliomyelitis acuta im Kindesalter.

Die folgende tabellarische Zusammenstellung wird das Alter der Patienten und Verbreitung der Lähmungen bei Beginn der Krankheit und der Behandlung zeigen.

Ausserdem wird dieselbe eine Übersicht über die Ergebnisse der Behandlung und deren Dauer geben.

Die beigefügten Photographien zeigen 2 ausgesprochene Fälle von der nicht häufigen Lähmung der ganzen Muskulatur des Rumpfes, kompliciert mit Lähmung beider Unterextremitäten. Die Röntgenphotographien I–IV zeigen die Entwickelung der Knochen der gelähmten Extremitäten im Vergleich zu denen normaler Individuen.

Endlich zeigt Röntgenphotographie Nr. V die Unterschiede in der Entwickelung der Knochen auf der gelähmten und gesunden Seite bei demselben Individuum.

[1] a. a. O., S. 48.

Geschlecht.	Beginn der Krankheit.	Alter beim Beginn der Krankheit.	Sitz der initialen Lähmungen.	Beginn der Behandlung.	Alter beim Beginn der Behandlung.	Zeit vom Beginn der Krankheit bis zu dem der Behandlung.	Lähmungen beim Beginn der Behandlung.	Datum der Entlassung.	Dauer der Behandlung.	Ergebniss der Behandlung.
1. Knabe	1887	14 Mon.	Beide Unterextremitäten.	Juni 19, 1893	7 Jahre	6 Jahre	Lähmung und theilweise Atrophie des linken Beines mit Pes varus; Parese des rechten Beines.	Sept. 7, 1893	80 Tage	20. Juni. Redressement, Gipsbandage. 8. Aug. Schienenstiefel. 14. Aug. Massage und Heilgymnastik. Bedeutende Besserung. Kann mit Leichtigkeit gehen. Gewicht bei der Aufnahme: 21.500. Gewicht bei der Entlassung: 22.710.
2. Mädchen	1888	1 Jahr	Unbekannt.	Juni 19, 1898	11 Jahre	10 "	Kyphose und linksseitige Scoliose. Linke Unterextremität gelähmt und atrophisch; Kontraktur in Knie- und Fussgelenk.	Juli 18, 1888	394 "	Massage und Heilgymnastik. Entlassen mit Hessings Corset, combiniert mit Schienenbandage für's linke Bein; hiermit geht Patient gut sowohl auf Treppen als zu ebener Erde. Gewicht bei der Ankunft: 23.800. Gewicht bei der Entlassung: 25.200.
3. Knabe	Sommer, 1889	6 Monate	Alle 4 Extremitäten.	Jan. 28, 1893	4 Jahre	3½ "	Linke Unterextremität gelähmt, atrophisch.	Febr. 19, 1895	752 "	Massage und Heilgymnastik. Völlige Funktionsfähigkeit des linken Beines. Gewicht bei der Ankunft: 11.200. Gewicht bei der Entlassung: 13.400.
4. Knabe*	August, 1889	16 Mon.	Rumpf und alle 4 Extremitäten.	Jan. 28, 1893	6 Jahre	4½ "	Beide Unterextremitäten gelähmt, atrophisch und mit starken Kontrakturen. Bedeutende rechtsseitige Scoliose. Parese in den Armen.	Oct. 27, 1894	637 "	Massage und Heilgymnastik. Hessings Corset. Unbedeutende Wirkung der Behandlung. Die Kontrakturen weniger ausgesprochen. Die Arme sind kräftiger, so dass Pat. Arbeit (Korbflechten) damit ausüben kann. Gewicht bei der Ankunft: 13.490. Gewicht bei der Entlassung: 14.850.

* Vergl. die beigefügte Photographie No. 1 (aufgenommen 1893) und Röntgenphotographie No. 1 (aufgenommen 1899). (Kontrolphotographie der Unterextremitäten eines gesunden

5. Mädchen†	Sommer, 1890	3 Jahre	Unbekannt.	Sept. 14, 1898	11 Jahre	8 Jahre	Die Unterextremitäten gelähmt. Flexionskontraktur in beiden Hüftgelenken. Valgusstillung beider Kniegelenke und des rechten Fussgelenkes. Lähmung der Körpermuskulatur, Scopulae alotae. Starke Lordose und linksseitige Scoliose.	Fortwährend in der Behandlung in der Klinik.		Massage und Heilgymnastik. Bedeutend mehr Rührigkeit in den Unterextremitäten und der Körpermuskulatur. Gewicht bei der Aufnahme: 23.000. Gewicht am 31. Juli, 1899, 31.700.
6. Mädchen	1890	4 Monate	Beide Unterextremitäten.	März 18, 1896	7 Jahre	6½ "	Lähmung und Atrophie der rechten Unterextremität. Pes varus et cavus. Die Zehen verkrüppelt.	April 17, 1896	30 Tage	Massage und Heilgymnastik. Wird mit Schienenstiefel völlig funktionsfähig entlassen. Gewicht bei der Aufnahme: 16.500. Gewicht bei der Entlassung, 18 800.
7. Knabe	1891	1 Jahr	Rechter Arm.	Jan. 9, 1894	4 Jahre	3 "	Lähmung und Atrophie der ganzen Oberextremität.	Juli 9, 1894	179 "	Massage und Heilgymnastik. Entlassen mit Schüsslers Bandage—ohne besondere Veränderung des Zustandes. Gewicht bei der Aufnahme: 13.490. Gewicht bei der Entlassung: 14.850.
8. Knabe	Herbst, 1892	15 Mon.	Beide Unterextremitäten.	Aug. 24, 1893	2¼ Jahre	1 "	Lähmung und Atrophie der beiden Unterextremitäten. Pedes aquino-vari.	Febr. 24, 1894	189 "	Massage und Heilgymnastik. Aktive Beweglichkeit der beiden Unterextremitäten. Die Kontraktur gehoben. Gewicht bei der Aufnahme: 9.620. Gewicht bei der Entlassung: 10.950.
9. Knabe	Herbst, 1892	3 Jahre	Beide Unterextremitäten.	Nov. 7, 1893	4 Jahre	1 "	Lähmung und Atrophie der rechten Unterextremität.	Juni 7, 1894	209 "	Massage und Heilgymnastik. Er geht gut und sicher auf dem rechten Bein. Gewicht bei der Aufnahme: 16.940. Gewicht bei der Entlassung: 18.350.

† Vergl. Photographie No. 2 und Röntgenphotographie No. III. (Kontrolphotographie der Unterextremitäten eines gesunden Mädchens von gleichem Alter, No. IV.)

Geschlecht.	Beginn der Krankheit.	Alter beim Beginn der Krankheit.	Sitz der initialen Lähmungen.	Beginn der Behandlung.	Alter beim Beginn der Behandlung.	Zeit vom Beginn der Krankheit bis zu dem der Behandlung.	Lähmungen beim Beginn der Behandlung.	Datum der Entlassung.	Dauer der Behandlung.	Ergebniss der Behandlung.
10. Knabe	1894	2 Jahre	Alle 4 Extremitäten und die Körpermuskulatur. Schwierigkeit beim Sprechen.	Sept. 19, 1898	6 Jahre	4 Jahre	Lähmung der Körpermuskulatur mit linksseitiger Scoliose. Der Kopf ruht gegen die rechte Schulter.	Dec. 19, 1898	92 Tage	Massage und Heilgymnastik. Pergament-Corset. Bedeutende Besserung. Der Kopf wird aufrecht gehalten. Gewicht bei der Ankunft: 15.000. Gewicht bei der Entlassung: 16.000.
11. Knabe	Mai, 1894	$2\frac{1}{2}$ "	Rechte Unterextremität.	Sept. 1, 1894	3 Jahre	6 Monate	Rechte Unterextremität gelähmt und atrophisch.	Sept. 10, 1894	10 "	Diphtherie. Uebergeführt nach Ullevolds Epidemie-Lazareth.
12. Knabe	Septemb., 1895	$3\frac{3}{4}$ "	Beide Unterextremitäten.	Jan. 14, 1896	4 Jahre	3 "	Linke Unterextremität gelähmt und atrophisch.	Jan. 15, 1896	1 Tag	Keuchhusten. Uebergeführt in die Nerven-Abtheilung.
13. Knabe ‡	Mai, 1896	1 Jahr	Rechte Unterextremität.	März 21, 1890	4 Jahre	3 "	Rechte Unterextremität gelähmt und atrophisch. Rechter Unterschenkel ist kürzer und weniger entwickelt als der linke. Länge des rechten Unterschenkels 16 Ctm. Länge des linken Unterschenkels 18 Ctm. Umfang des rechten Unterschenkels 13 Ctm. Umfang des linken Unterschenkels 18 Ctm.	Juli 8, 1899	110 Tage	Massage und Heilgymnastik. Schienenbandage, womit er sicher und gut geht. Gewicht bei der Ankunft: 11.400. Gewicht bei der Entlassung: 12.500.
14. Knabe	Juli, 1896	11 Mon.	Beide Unterextremitäten.	Dec. 7, 1896	17 Mon.	6 "	Rechte Unterextremität gelähmt und atrophisch.	Dec. 27, 1896	20 "	Starb an Bronchopneumonie.

‡ Vergl. Photographie No. 3 und Röntgenphotographie No. V.

15. Knabe	August, 1896	2 Jahre	Rechte Oberextremität.	Jan. 8, 1897	$2\frac{1}{2}$ Jahre	6 Monate	Rechte Oberextremität gelähmt und atrophisch.	Febr. 15, 1897	38 Tage	Massage und Heilgymnastik. Der Arm ist funktionsfähig, so dass er ihn bewegen und Gegenstände halten kann. Gewicht bei der Ankunft: 11.900. Gewicht bei der Entlassung: 12.600.
16. Knabe	August, 1896	15 Mon.	Rechte Unterextremität.	Nov. 6, 1896	$1\frac{1}{2}$ Jahre	3 "	Rechte Unterextremität gelähmt und atrophisch.	Nov. 13, 1896		13. Nov. Scharlachfieber. Uebergeführt ins Ullevold Epidemie-Lazareth; starb am 18. Nov. 1896.
17. Knabe	August, 1896	11 "	Rumpf, linke Oberextremität, beide Unterextremitäten.	Sept. 11, 1896	1 Jahr	1 Monat	Körpermuskulatur u. linke Unterextremität gelähmt und schlaff.	Sept. 17, 1896		Scharlachfieber. Uebergeführt ins Ullevold Epidemie-Lazareth.
18. Knabe	August, 1896	4 Jahre	Rumpf und alle 4 Extremitäten.	Nov. 4, 1896	$4\frac{1}{4}$ Jahre	3 Monate	Die Schulterpartien und die Oberarme sowie rechte Unterextremität gelähmt und atrophisch. Pes equino-varus dexter.	Juli 6, 1897	234 "	Massage und Heilgymnastik. 25. Dec. 1896: Pat. kann ohne Stütze stehen. 10. März 1897: Pat. kann heute gehen, indem er sich an's Bett stützt. 11. April 1897: Pat. ist gestern allein die Treppe hinaufgegangen. 19. Apr. 1897: Pat. ist den ganzen Tag auf und springt umher und spielt mit den anderen Kindern. Gewicht bei der Aufnahme: 16.000. Gewicht bei der Entlassung: 18.500.
19. Mädchen	Septbr., 1896	9 Monate	Beide Unterextremitäten.	Jan. 4, 1897	1 Jahr	3 "	Lähmung und Atrophie beider Unterextremitäten.	März 11, 1897	56 "	Massage und Heilgymnastik. Bedeutende Besserung. Pat. kann spontan beide Beine bewegen, besonders das rechte. Gewicht bei der Aufnahme: 8.500. Gewicht bei der Entlassung: 8.500.

Geschlecht.	Beginn der Krankheit.	Alter beim Beginn der Krankheit.	Sitz der initialen Lähmungen.	Beginn der Behandlung.	Alter beim Beginn der Behandlung.	Zeit vom Beginn der Krankheit bis zu dem der Behandlung.	Lähmungen beim Beginn der Behandlung.	Datum der Entlassung.	Dauer der Behandlung.	Ergebniss der Behandlung.
20. Knabe	Septbr., 1897	4 Monate	Alle 4 Extremitäten.	Aug. 4, 1898	15 Mon.	11 Monate	Linker Arm und beide Unterextremitäten gelähmt und atrophisch.	Jan. 11, 1897	161 Tage	Pat. konnte nicht mit Massage behandelt werden, da er nach jedem Versuch damit grosse Schmerzen und Temperatur-Steigerungen bekam. Gewicht bei der Aufnahme: 9.800. Gewicht bei der Entlassung: 9.800.
21. Mädchen	August, 1897	9 Jahre	Unbekannt.	Aug. 27, 1897	9 Jahre	Einige Tage	Rechte Unterextremität gelähmt und atrophisch.	Mai 4, 1898	250 "	Massage und Heilgymnastik. Das Bein kann gut und leicht gebraucht werden.
22. Mädchen	Decbr., 1897	3 "	Rumpf und Unterextremitäten. Näselnde Aussprache.	Jan. 21, 1898	4 Jahre	1 Jahr	Rumpf und Unterextremitäten gelähmt und atrophisch.	März 18, 1898	56 "	Massage und Heilgymnastik. 9. Febr. 1898: Pat. geht jeden Tag im Gängelkorb. 25. Febr. 1898: Pat. geht gut ohne Stütze, springt umher und spielt mit den anderen Kindern. Gewicht bei der Aufnahme: 12.000. Gewicht bei der Entlassung: 13.200.
23. Mädchen	Unbekannt		Beide Unterextremitäten.	Dec. 9, 1893	7 Jahre		Linke Unterextremität gelähmt und atrophisch. Tibia nach hinten subluxit. Pes equinovarus.	Mai 22, 1894	164 "	Massage und Heilgymnastik. Bedeutende Besserung. Pat. geht gut und sicher auf beiden Beinen. Gewicht bei der Aufnahme: 16.940. Gewicht bei der Entlassung: 18.350.

PHOTOGRAPHIE No. 1 (1893).

Sechs jähriger Knabe mit Lähmungen der Musculatur des Rumpfes und der Unterextremitäten nach Poliomyelitis.

PHOTOGRAPHIE No. 2 (1898).

Elf jähriges Mädchen mit Lähmungen der Musculatur des Rumpfes und der Unterextremitäten nach Poliomyelitis.

PHOTOGRAPHIE No. 3 (1899).

Vier jähriger Knabe mit Lähmung und Atrophie der Unterextremitäten nach Poliomyelitis.

Röntgenphotographie No. 1 (1899).

Dreizehn jähriger Knabe (vergl. Photographie No. 1 und No. 4 der Tabelle) Sagittalphotographie der beiden Unterextremitäten.

Expositionszeit $3\frac{1}{2}$ Minuten. Strahlenstärke No. 11. Röhrenabstand 50 cm.

Röntgenphotographie No. 2 (1899).

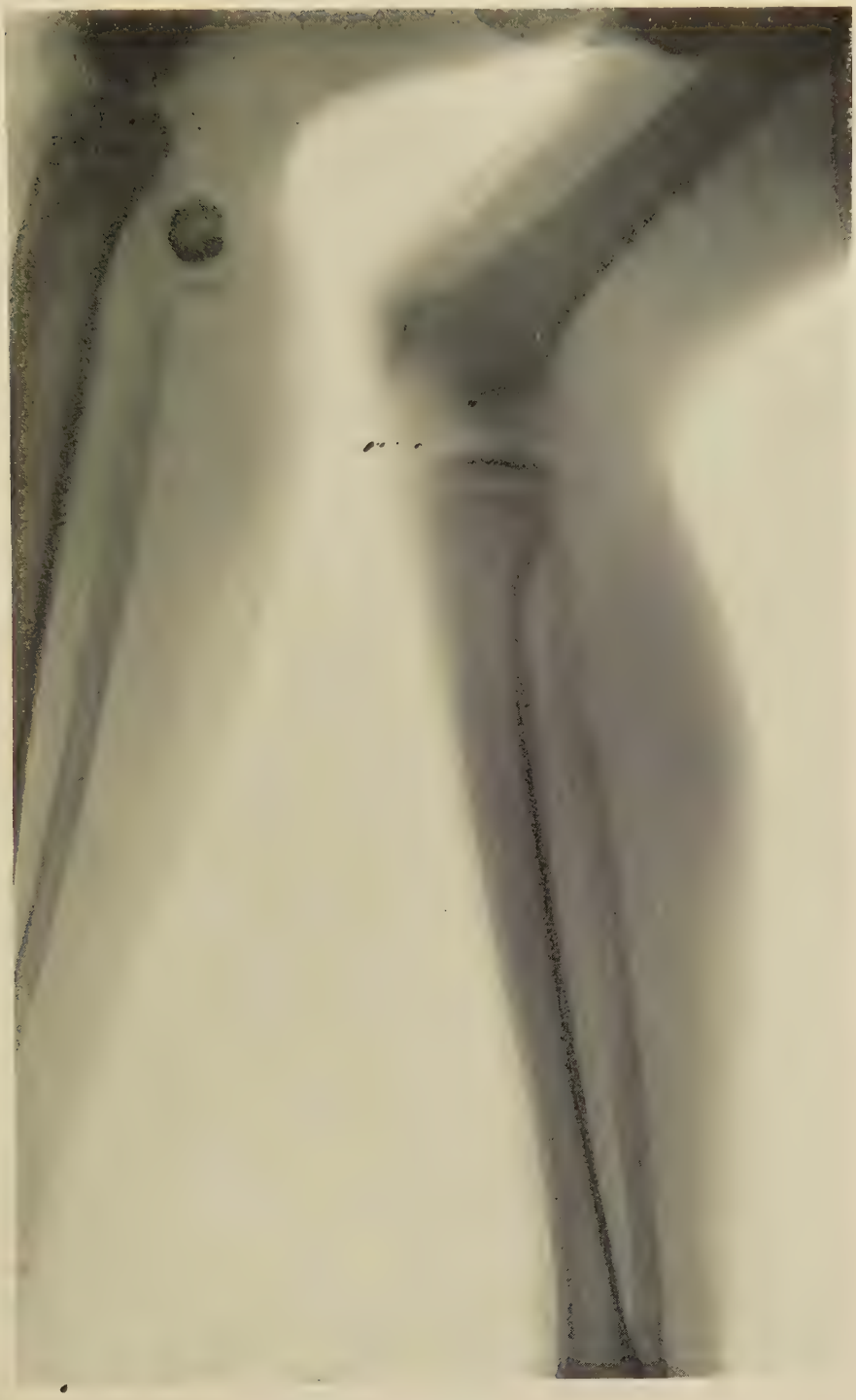
Kontrolphotographie eines gesunden dreizehn jährigen Knabens.

Expositionszeit $5\frac{1}{2}$ Minuten. Strahlenstärke No. 12. Röhrenabstand 50 cm.

Röntgenphotographie No. 3 (1899).

Elf jähriges Mädchen (vergl. Photographie No. 2 und No. 5 der Tabelle) Sagittalphotographie der beiden Unterextremitäten.

Expositionszeit 4 Minuten. Strahlenstärke No. 11. Röhrenabstand 50 cm.



Röntgenphotographie No. 4 (1899).

Kontrolphotographie eines gesunden elf jährigen Mädchens.

Expositionszeit 5 Minuten. Strahlenstärke No. 12. Röhrenabstand 50 cm.

Röntgenphotographie No. 5 (1899).

Vier jähriger Knabe mit Lähmung der linken Unterextremität. (Vergl. No. 13 der Tabelle.)
Frontalphotographie der beiden Unterschenkel.

Expositionszeit 2½ Minuten. Strahlenstärke No. 12. Röhrenabstand 35 cm.

Untersucht man nun die Ergebnisse der Behandlung, so wird man sehen, dass 4 Patienten nach einem Aufenthalt von wenigen Tagen in der Klinik, als an epidemischen Krankheiten leidend, entlassen wurden. 1 starb on Bronchopneumonie nach 20-tägiger Behandlung. Bei der Sektion fanden sich die gewöhnlichen Veränderungen.

Die übrigen Patienten wurden alle mit *Heilgymnastik* und *Massage* behandelt. Bei einzelnen wurden auch warme Fichtennadelbäder angewandt. Elektrische Behandlung kam während des Aufenthalts in der Klinik bei keinem Patienten zur Anwendung; aber bei einzelnen war dieselbe früher ohne Nutzen versucht worden. Die mitgeteilten Fälle können daher als ein Beitrag zur Frage über die Bedeutung der Massage und Heilgymnastik für die Behandlung der Poliomyelitis anterior acuta verwendet werden.

Unter den 18 Fällen, bei denen diese Behandlung verordnet wurde, war ein 15 Monate alter Knabe, bei dem dieselbe nicht angewandt werden konnte, da sie sehr grosse Schmerzen und Temperatursteigerungen verursachte.

Von den übrigen 17 verliessen 11 die Klinik mit vollständig brauchbaren Gliedern; in 3 Fällen mit Schienenstiefel. In 2 Fällen war die Besserung ganz bedeutend, wenngleich die Patienten nicht in vollem Gebrauche ihrer Glieder waren.

In 4 Fällen war die Besserung unbedeutend oder nicht nachweisbar. In den Fällen, wo die Patienten mit Brauchbarkeit der gelähmten Körperteile entlassen wurden, hatte die Lähmung ihren Sitz:

In beiden Unterextremitäten	in 2 Fällen
" der rechten Unterextremität	" 4 "
" " linken "	" 2 "
Im Rumpf und beiden Unterextremitäten	" 1 Fall
" " " in der linken Unterextremität	" 1 "
In Schulter, Oberarm und linken Unterextremität	" 1 "

Bedeutende Besserung trat in 1 Fall von Lähmung des Rumpfes und in 1 Fall von Lähmung von Rumpf und Unterextremitäten ein.

Unbedeutende oder keine Besserung wurde beobachtet:

in 1 Fall	von Lähmung des Rumpfes und aller 4 Extremitäten.
" 2 Fällen "	" der rechten Oberextremität.
" 1 Fall "	" " beiden Unterextremitäten.

Der Erfolg der Behandlung schien nicht von der Zeit, die zwischen dem Auftreten der Krankheit und dem Beginn der Behandlung verstrichen war, abhängig zu sein.

Es zeigte sich nämlich, dass in den Fällen, die mit bedeutender Besserung entlassen wurden, die Behandlung von einigen Tagen bis 10 Jahren nach Anfang der Krankheit begonnen hatte; in den Fällen, wo

die Behandlung geringe oder gar keine Wirkung hatte, hatte dieselbe ½ Jahr bis 4½ Jahre nach Beginn der Krankheit angefangen.

Unseren Erfahrungen nach scheint jedoch kein Bedenken vorhanden zu sein, die Behandlung sofort, nachdem das akute Stadium vorüber ist, zu beginnen. Es scheint im Gegentheil, als ob die Behandlung kürzere Zeit erfordert und von vollkommenem Erfolg gekrönt ist, wenn dieselbe zeitig eingeleitet wird.

Ausserdem wird dadurch dem Entstehen von Kontrakturen und Schiefstellungen vorgebeugt.

Die Dauer der Behandlung war sehr verschieden.

Dieselbe zeigte in einigen Fällen in kürzerer Zeit, z. B. in 3 Monaten, ein gutes Resultat, während bei anderen ½ bis 2 Jahre gearbeitet werden musste, ehe die günstigen Wirkungen sich einfanden.

In den Fällen, wo kein Erfolg erzielt wurde, hatte man die Hoffnung nicht aufgegeben, ehe man von ½ bis fast 2 Jahre mit den Patienten gearbeitet hatte.

Die Behandlung wird in der Klinik nach den Grundsätzen angeführt, welche die Ausgangspunkte für die schwedische Heilgymnastik bilden, und die in Dr. Wide's Buch: *Handbuch der medicinischen Gymnastik*, 1897, dargestellt sind.

In der Hauptsache geht man bei uns nach folgenden Regeln vor:

Die Behandlung einer Poliomyelitis muss energisch sein und täglich ausgeführt werden—am liebsten bis der Körper des Patienten völlig entwickelt ist. Gleichzeitig muss derselbe gut ernährt und weder geistig noch körperlich überangestrengt werden.

Die Behandlung beginnt mit *Massage*, die wiederum in zwei Abtheilungen zerfällt:

Effleurage, die aus Streichungen besteht, die mit der flachen Hand ausgeführt wird und *Petrissage*, die in Walken der Muskeln mit den Fingerspitzen unter drehender, vorwärts führender Bewegung besteht.

Diese beiden Handgriffe müssen sehr leicht sein und abwechselnd 5—10 Minuten zur Zeit ausgeführt werden. Die Massagebehandlung variiert von 10—20 Minuten, je nach Alter und Entwicklung der Patienten.

Da die Patienten in der Regel keine Bewegung mit dem paralytischen Gliede vornehmen können, wird vollständig passive Gymnastik, Rollen, Drehungen, Beugungen, und Streckung erteilt. Diese Bewegungen dauern von 10 bis 15 Minuten.

Die ganze Behandlung darf in den ersten Monaten ½ Stunde nicht überschreiten. Sobald es dem Pat. möglich, muss er bei den Übungen mitarbeiten und selbst die verschiedenen Bewegungen vornehmen. Hierbei wird die Massage durch *Tapotement* verstärkt, die in Hacken

der Muskelpartien mit gespreizten Fingern und losem Handgelenk und *Nervenstreckung*, die aus kurzen, starken Drückungen auf die grossen Nervenstämme besteht.

Hat der Patient angefangen, aktive Bewegungen auszuführen, so geht man zu den sogenannten *Widerstandsbewegungen* über, wobei Bewegungen unter Widerstand seitens des Gymnasten oder Patienten vorgenommen werden. Dieser Widerstand darf jedoch nicht stärker sein, als dass der Patient ihn mit gutem Willen überwinden kann.

Es werden nun zur früheren Behandlung folgende Übungen hinzugefügt: *Streckhängende Beinteilungen*, wobei der Patient in einem Trapez oder dergl. hängt; der Gymnast fasst um die Beine in der Mitte der Waden. Der Patient führt beide Beine auswärts, und wieder zurück unter Widerstand von Seite des Gymnasten, worauf dieser die Bewegung ausführt, während der Patient Widerstand leistet.

Hochstützstehende Bein-Rückwärts-Ziehung mit Widerstandsbewegung: Der Patient stellt sich auf ein Taburett und stützt sich mit den Händen gegen den Sprossenmast. Das gelähmte Bein wird nun rückwärts und vorwärts geführt bei abwechselndem Widerstand von Gymnast und Patient.

In Betreff der Oberextremitäten werden Armrollen, Beugungen und Ziehungen mit abwechselndem Widerstand ausgeführt.

Ausserdem *Rückenstützstehende doppelte Plan-Arm-Führung*, welche darin besteht, dass der Patient gegen eine Wand gestellt wird mit einem Kissen im Rücken, so dass der Brustkasten vorgeschoben wird. Der Gymnast stellt sich nun vor den Patienten und fasst um sein Handgelenk, worauf der gestreckte Arm von der Mitte seitwärts ganz bis zur Wand geführt wird mit abwechselndem Widerstand.

Es ist selbstredend, dass Deformitäten sehr genau behandelt werden müssen. Oft ist Scoliose vorhanden, die specielle Aufmerksamkeit erfordert. Die Muskulatur des Rückens muss nach den oben gegebenen Regeln behandelt werden. Ausserdem wird Streckung im Galgen und sitzender Seitenbeugung angewendet, die darin besteht, dass der Patient mit fixiertem Becken sitzt und den Oberkörper nach der konvexen Seite beugt, während der Gymnast einen kräftigen Gegendruck giebt.

THE AFFERENT ELEMENT IN EPILEPSY.

By WILLIAM H. THOMSON, M.D., LL.D.,

Physician to Roosevelt Hospital; Late Professor of the Practice of Medicine, New York University Medical College; and Physician to Bellevue Hospital.

TO arrive at a correct appreciation of the essential nature of epilepsy, it is necessary first of all to settle what element in it is invariable. This is because with no disease is its definition so little deducible from its symptoms as in epilepsy, for however pronounced these symptoms often may be, yet they differ so widely in different cases, or at different times in the same case, that little aid can be derived from them as to the true nature of the affection. Thus, most writers speak of epilepsy as beginning with "a discharge" of nervous force in some cortical centre or area, the conception evidently being of something like the electrical discharge of a Leyden jar or of the explosion of some unstable chemical compound. But I venture to say that this conception is mainly derived from the striking motor accompaniments of a convulsive paroxysm, but if attention were equally directed to the symptoms accompanying true epileptic attacks which are not at all convulsive, this idea of "explosion" would be as little suggested as in a case of syncope.

Again, one of the most pronounced symptoms is the loss of consciousness. But this accompaniment also is not invariable and hence does not hold an essential or constant relationship to the disease. It is doubtful whether it has anything in common with the mechanism of sudden loss of consciousness from a blow on the head, or in apoplexy, and still less in syncope. In fact, loss of consciousness *per se* is the least helpful of all symptoms towards affording a clue to any cortical process, as the problem of common sleep testifies. In some epileptics, on the other hand, the affection of the consciousness suggests a process of inhibition or of abeyance of function rather than of stimulation, as the chief factor in the initiation of the symptoms. Thus a patient of mine, a very intelligent gentleman, stated that his attacks always began with an instantaneous aphasia which his description

proved to be of the motor kind. This aphasia would commonly last for an hour or more, during which time he would have such perfect command of his consciousness that he made a careful experimental study of his experience before the onset (which hitherto had been invariable) of loss of consciousness and convulsions. Once the aphasia came on while on deck of a German steamer crossing the Atlantic. He at once went to his stateroom, remembering the while that he had inconveniently packed his writing materials at the bottom of his trunk. When he got them he began a note to send by the steward to the ship's surgeon, but though he was proficient in French and German, owing to his early education at school in both countries, he found that he could not finish the sentence which he began in German, so he began another in French, but when he found his French also leaving him he fell back on his native English, to find that this also broke off in the same fashion as in the other two languages. Being an excellent performer on the violin, he experimented with that during his aphasic attacks and found that he could both play well by ear and recognize when he "flatted" at certain notes. I am very sorry that I neglected to ask him whether he could read music at these times.

In fact, there is only one element which is invariable in epilepsy whatever the other developments be, and hence it deserves the most attention as the nearest related of all to the true pathology of the disease. That element is suddenness, or in other words its beginning is instantaneous. Epilepsy is the only sudden disease. Other diseases may be rapid in their onset, but none of them, including nervous diseases as well, are sudden in the fashion that epilepsy constantly is. This pathognomonic suddenness of course refers only to the first step in the morbid series. The subsequent train of symptoms may be quite gradual in their development, as in our patient just described, but it would have been epilepsy all the same, and nothing but epilepsy if he had had attacks of only momentary aphasia and nothing further, as was actually the case after he came under my treatment. The only reason why this essential feature of epilepsy is not duly recognized is because if certain prominent common symptoms do not accompany the attack it is rated as "incomplete" epilepsy. But a case of long-continued attacks of petit mal is certainly not a patient with "incomplete" epilepsy, nor any easier to cure than the "complete" epileptic is, and no more warrants such a minimizing term than it would be to pronounce a patient with only a few variolous pustules as a case of incomplete smallpox. So no epileptic is free from his disease if he has occasionally some really sudden nervous symptom,

for every sudden nervous symptom periodically recurring is epileptic, though it seems to be but a momentary sensory or motor disturbance. The only exceptions which we are inclined to admit to this dictum are in some tabetic lightning pains, and some cases of sudden abasia, but their connections serve to exclude them.

What does this specific suddenness imply? At first sight it seems to lend support to the theory of a primary explosive discharge of nerve force. There is, however, another way of interpreting it, which appears to me to be in closer accordance with the fundamental elements of nervous function. The stones which compose an arch bear a mutual relation to each other in the distribution of the constant force of gravitation, which acts upon them from the time when they were built together. Loosening one of the stones would be the occasion of an instant and great disturbance, but one which could hardly be called the result of an explosion, or still less of a stimulation. Or to take a different illustration. On board an ocean steamer I was reading on the sofa in the cabin just above the propeller. While passing over a large wave the propeller was lifted out of the water with the effect of causing the whole vessel to be violently shaken as by a convulsion. The propeller had no new force communicated to it. What occurred to it and to the vessel was solely the effect of the sudden withdrawal of the normal "inhibition" of the water in which the propeller revolved, and against which inhibition or resistance it was constructed to act.

Analogous to these illustrations of marked disturbances caused by sudden derangements in a regulated balance of resistance between parts, we think that we may look in the direction of derangement of normal mutual inhibition between nerve centres for the true process of epileptic phenomena. Such a conception calls for no spontaneous explosion or stimulation of a nerve centre, but simply for a suspension of its normal relations to other centres. For the more the origin of the relation of nerve centres to each other is studied, the plainer appears the necessity of the existence of a constant regulative mutual inhibition between them for the performance of their normal functions.

Now the beginning of every nervous action is always on the afferent side. A spontaneous, *i. e.*, a primary, motor or efferent nervous discharge is unknown in physiology. Why should it occur without an afferent excitation in epilepsy? If any nervous discharge be regarded as explosive, still it is by some afferent train that the explosion is always lit. All efferent phenomena are in response to afferent excitation and to nothing else. Excluding for the present any metaphysical questions of action by the will being spontaneous, we may say that for all other efferent activity, however continuous or complicated, the

necessary antecedent is afferent excitation of some kind, however slight or single it be. The motor nerves of over a hundred muscles have to co-operate to execute the convulsive act of sneezing, but they all move in successive response to the afferent stimulus from a twig of the trigeminus.

The second point which we would make is that originally all nervous functions were organized by repeated afferent excitation. In other words, a habitual response to the same recurrent afferent stimulus ends in a definite mode of action which finally constitutes a specific function. It is by the constant repetition of the same recurring excitation that certain groups of neurons become disciplined to react uniformly to a certain, definite afferent impression, originally reaching one of them and then habitually passing to others in association with it. But this in turn implies the establishment of a definite reaction of the nerve centres upon one another in the form of a regulated inhibition of all irregular response to afferent excitation. Without such mutual inhibition there would be no co-ordination, and thus finally no specific function. Afferent habit being the original organizer of the nervous system, the latest acquired cerebral functions are the least organized, because they have the shortest history of habit. The oldest are the most organized, so that they seem to act automatically because they are habituated the longest to their afferent and efferent ways. But inhibition in turn implies the presence and accumulation of nerve force to inhibit. A nerve centre so functionally constructed, implies a mutual interaction quite analogous to that between the stones composing an arch. These are constructed to resist the impact of a great pressure acting vertically, while a much less greater impact acting horizontally, to resist which they were not constructed, might easily overthrow them.

The first inference which we would draw from these principles as applicable to our subject is this: that any really new and unaccustomed afferent excitation reaching an organized nerve centre may be dangerous, by its deranging the habitual reactions of the different elements of that centre. For example, the nuclei along the floor of the fourth ventricle are the most confirmed in their afferent and efferent habits of any centre in the body. Such an unprecedented excitation, therefore, as that caused by the first passage of a stomach tube, followed by a gastric douche, never came to them before. Now it may happen, for it often has happened, that this wholly new afferent stimulus leads to spasm of the muscles of the throat and neck first, then to general tetanic rigidity of the body, or, in time, to violent convulsions, ending sometimes in a veritable status epilepticus and death. Just the

same epileptiform convulsions, likewise ending fatally in a number of recorded instances, have followed irrigation of the pleural cavity. In both these instances the one element they have in common as an antecedent to the severe motor disturbance is a wholly new, or, so to speak, entirely strange afferent stimulus.

On the other hand, some poisons or toxines circulating in the blood seem to cause convulsions by affecting primarily the peripheral sensory nerves. In poisoning with strychnia, as well as in tetanus and in hydrophobia, the lightest surface touch, or a breath of air, influences the motor response of spasm. It would seem as if perfect peripheral quiet, so to speak, would then prevent central explosion. This phenomenon is quite as explicable on the supposition that the derangement is due to morbid peripheral, as to morbid central, excitability. Because if it be maintained that the convulsions are due to morbid excitability of the nerve centres, the surface excitability would have still to remain unexplained, except by the clumsy hypothesis that we all would be liable to convulsions from a breath of air but for our central stability. It would seem easier to suppose that the action of the poison altered the afferent stimuli so as to deprive them of their normal character altogether, with a corresponding alteration in the effect of their impressions on the centres.

Be that as it may, the clinical fact of suddenness in the first process in epilepsy points to a derangement so immediate in the relations of nerve centres that the conception of its being essentially a disturbance of a habitual equilibrium like that of the arch in our illustration, is much more probable than the conception of a spontaneous chemical explosion of efferent activity. Ordinary muscular tonus is an example of constant efferent response to constant afferent impression which may illustrate the normal status of interrelation between nerve centres generally. Such a thing as an independent, self-acting efferent centre is non-existent. An irregularly acting motor centre has been irregularly excited, rather than self-excited, and the natural direction to look for the source of its disturbance is in what alone excites a motor centre, namely, an afferent stimulus.

On these lines my definition of epilepsy would be this: *Epilepsy is a disease characterized by a sudden derangement of the normal regulative inhibition existing between cortical nerve centres, induced in the first instance by an abnormal afferent excitation.*

This statement of the pathology of epilepsy shifts the *primary* seat of the disease from the motor or efferent to the sensory or afferent portions of the nervous structures involved. It also follows that, as all normal efferent activity is the outcome of habitual response to

accustomed afferent excitation, so a frequently recurring abnormal afferent impression will end in causing a corresponding abnormal response in the efferent centres. An instability of these centres will thus become established. The liability to epilepsy being confirmed by mere habit is therefore only what we would expect from the rôle of afferent habit in nervous functions generally. Hence, the relative instability of the cortical centres in epileptics does not show that the disease began with or was due to such instability, but rather would indicate that the insta bility is itself the result of an antecedent perversion in some afferent tract, which may have had a sudden or a very gradual development; for we have many indications that the effects of afferent excitation may at first be slowly cumulative. Once induced, however, subsequent repetition of the same effect may follow upon much slighter excitation.

The second point we would maintain is that the afferent origin, so to speak, of epilepsy is not invalidated by the development of the disease as a result of intercranial focal cortical irritation, such as by trauma, tumor, etc. Every cortical efferent centre can be likewise afferent to other centres, for in that fact lies all association between different centres. That this is so seems demonstrated by the phenomenon of auras. These of course are sensory phenomena, and what is more, they always precede and do not follow the efferent symptoms. That these sensory manifestations sometimes apparently begin in the extremities, while the focus of irritation is within the cranium, is no proof that the process is starting as a "discharge" in the motor area, but rather is equally explicable as an intercranial excitation referred to the periphery in the same fashion that irritation of a sensory nerve anywhere along its course is also referred to its periphery.

On the other hand, those cases termed reflex epilepsy, because they appear to be induced by a distinct irritation, such as intestinal worms, renal calculus, etc., are, according to our view, not a class by themselves, simply because every case of epilepsy is equally reflex and never the result of a spontaneous central discharge.

Opinions on the pathology of a given disease have only an academic interest, unless they lead to practical results either in prophylaxis or in treatment. It seems to me that greater attention to the part which abnormal afferent excitation may play in epilepsy, rather than to supposed central instability, will often result in more successful management of the complaint. The demonstrated action upon the animal economy of some of our most reputed remedies for epilepsy apparently confirms this surmise. Thus, bromides, according to a wide consensus of experimenters, act on the peripheral sensory apparatus exclusively,

when administered to animals in doses corresponding to the therapeutic doses in man. Under their influence, a frog or a rabbit may have its reflex activity wholly abolished, and even show cutaneous anæsthesia to be complete, and yet when alarmed they jump vigorously, thus proving that their cortical efferent functions are intact.

The peripheral sensory effect of the bromides can be heightened by the simultaneous administration of chloral and antipyrine, so that with their aid smaller doses of the bromides will reduce reflex excitability, and I have turned this fact to good account in treatment. I would recommend, therefore, a careful examination into the state of the afferent system, particularly of the chief viscera in every case of epilepsy. I am inclined to believe that three fifths of all cases originate in some branch of the great vago-glosso-pharyngeal afferent tract. Thus the most incessantly active area in the body for reflex activity is at the crossing of the tracts of respiration and of deglutition. Rapid eating and drinking keeps that nervous mechanism in a constant state of excitement, and I feel sure that I have met with cases in which the first attack was caused by the habitual hurry of the patients in this respect. The difficulty which we often encounter, especially in young epileptics, to induce them to swallow deliberately is an illustration of a morbid excitability of those parts, rather than of a merely acquired habit. Gastric auras and a bad breath, either before or after a fit, are indications which I never neglect, for a toxæmia from auto-infection supplies us too often with sensory derangements to doubt that the same agencies may in epileptics be the excitants of afferent derangement.

On the other hand, the transmission of an abnormal afferent impression frequently can be prevented or turned aside, so to speak, by an artificially induced counter-impression. Acting on this principle, I have for many years employed the red pepper pack at night to the whole surface, of an infusion of the strength of one half to one drachm of capsicum to the pint of water, and applied until the whole skin is reddened. At one of my clinics I had a very confirmed case of a boy aged ten, who had almost daily attacks. I told the class that to test the effects of this measure I would give no medicine but prescribe the pack, with directions to the mother to report at the end of the week on the number of fits. She did not return for five weeks, and explained her absence because he had not had a fit again until the previous day. Any measure which breaks up an afferent habit as such should be used as an adjuvant to the more continuous afferent sedation of the bromides.

The new conception of the elements of the neuron has done much

to displace from our minds the exclusive sway of the central nerve cell. It has likewise raised greatly our consideration for the peripheral expansion of the nervous system. End organs, without as well as within, rather than cells, now seem to be the chief factors in nervous function, and at any rate, in disease we can no longer minimize the share of the afferent division of the nervous arc.

EINE BETRACHTUNG ÜBER DIE ERNÄHRUNG DES KINDES JENSEITS DES SÄUGLINGSALTERS.

VON DR. OTTO HEUBNER,

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ZUR Ehrung eines Mannes, der viele Zeit seines erfahrungsreichen Lebens der Diaetetik des Kindes, besonders des Säuglings gewidmet hat, dürfte es nicht unangebracht sein, einige Worte über die Ernährung des gesunden Kindes zu sagen. Das Studium dieser Frage ist für den Arzt viel nothwendiger, als es heute noch von vielen jungen Adepten unserer Kunst angesehen wird. Denn unzählige Male wird er gerade hierüber von besorgten Müttern um Rath gefragt und vielleicht nicht sehr viel seltener wird er sich bei ernstlicher Ueberlegung sagen müssen, dass er seine Antwort zwar von allgemeinen physiologischen Grundsätzen, aber nicht von solchen Regeln beherrschen lässt, die auf einer speciellen Erforschung des Kindesalters in dieser Richtung beruhen.

Am Besten steht die Sache noch bei der Säuglingsernährung. Hier liefert uns Mutter Natur in der Ernährung an der Brust ein Beispiel, dessen möglichst getreue Nachahmung uns zum erwünschten Ziele gelangen zu lassen verspricht. Denn wir sehen, dass bei der natürlichen Ernährung eine völlig gesunde Funktion der Organe und ein ganz regelmässiges Wachsthum des Gesammtkörpers sich vollzieht — ein Ideal, über das wir nicht hinaus können. Dieses Beispiel ist nun aber durch zahlreiche Untersuchungen tüchtiger Forscher in einer solchen Genauigkeit nach qualitativer und quantitativer Beziehung studirt, dass es zur Richtschnur für unser Verhalten bei der künstlichen Ernährung sehr wohl genommen werden kann, wenn auch mit Kritik, sorgfältiges Mittelbilden aus der Menge von Einzelbeobachtungen und Anerkennung der Thatsache, dass auch noch manche Eigenschaften der Frauenmilch mangelhaft bekannt sind. Sodann liegen schon zur Zeit über die künstliche Ernährung des Säuglings eine so grosse

Anzahl zuverlässiger wissenschaftlicher Untersuchungen vor, dass diese Frage in immer wachsendem Maasse von Jahr zu Jahr zu grösserer Klarheit sich durchringt. Diese leiden alle freilich an einem sehr grossen Uebelstande: sie betreffen fast, oder eigentlich ganz, ausnahmslos nicht völlig normale Versuchsobjekte, sondern haben es bisher stets mit leichten, ja in der Mehrzahl der Fälle sogar mit beträchtlichen pathologischen Zuständen zu thun. So ist die wissenschaftliche Erörterung der künstlichen Ernährung des *gesunden* Säuglings in der That noch ein unerfülltes Erforderniss. Es ist aber auch sehr schwer abzusehen, wie ihm abgeholfen werden soll. Denn einmal ist kaum einzusehen, wie das erforderliche Material beschafft werden soll, andererseits ist es bei den uns zur Verfügung stehenden Untersuchungs-Methoden bisher nicht zu vermeiden gewesen, dass die ihnen unterworfenen Kinder, wenn alle Einnahmen und Ausgaben sorgfältig gemessen wurden, immer wenigstens leichte Störungen ihres Befindens, besonders ihrer Verdauung, erfuhren. Dafür liegen aber eine grosse Reihe brauchbarer, sorgfältig durchgeführter Einzelbeobachtungen über die allgemeinen Resultate künstlicher Säuglingsernährung vor, dass diese zur Aufstellung von Regeln benützt werden können, nach denen unser praktisches Verhalten sich mit Vortheil richten kann. Es ist sehr zu wünschen, dass diese Einzelbeobachtungen sich in immer höherem Grade vermehren. Ich möchte mich in dieser Beziehung besonders an die jungen Aerzte und deren Frauen wenden. Den letzteren, ich nenne die Damen Frau *Ahlfeld*, Frau *Haehner*, Frau *Feer* u. A., ist die paediatrische Wissenschaft für ihre höchst verdienstvollen gemeinschaftlich mit ihren Gatten ausgeführten unermüdlich fortgesetzten Wägungen während des Stillens zu dem grössten Danke verpflichtet. Nicht minder dankbar wäre es zu begrüssen, wenn recht zahlreiche Aerztefamilien ähnliche Beobachtungen über ihre *künstlich* ernährten Kinder mittheilen würden.

Anstalten dürften für derartige Beobachtungen schwerlich jemals das geeignete Feld darbieten. Das wird immer die Familie bleiben.

Immerhin darf schon jetzt behauptet werden, dass unser ärztliches Handeln in Bezug auf die Ernährung des Säuglings, gestützt auf das schon jetzt vorhandene wissenschaftliche Material, ein unverhältnissmässig sichereres und zielbewussteres ist, als das noch zu der Zeit der Fall war, als die älteren Mitarbeiter an dieser Festschrift ihren Studien oblagen.

In viel geringerem Maasse ist dies aber der Fall, wenn wir den Boden des Säuglingsalters überschreiten. Hier fehlt vor Allem das natürliche Beispiel, hier setzt die Kinderstubenerfahrung, ein sehr unsicheres Fundament, hier setzen aus der Physiologie der Erwachsenen

übertragene Vorstellungen, hier aber auch allerhand hypothetische Gedankengänge, Scheinbeweise, ja selbst ganz willkürliche Phantasien ein, um die Grundsätze festzustellen, die für die weitere Ernährung des Kindes maassgebend sein sollen.

Was nun an brauchbaren Unterlagen über den Gegenstand vorhanden, soll in den folgenden Zeilen dargelegt werden.

Betrachten wir zunächst die Zeit der Entwöhnung. Die allerdings noch wenig zahlreichen Messungen der Nahrungsmengen, welche den Säuglingen nach Absetzung von der Mutterbrust, in Gestalt von Kuhmilch oder anderen Nahrungsgemischen (Mehlsuppen mit Milch, condensirte Milch u. dgl.) gereicht wurden, haben ein auffallendes Resultat ergeben, auf das zuerst wohl *Forster* hingewiesen hat, das aber besonders *Wilhelm Camerer* betont hat.[1] Ich werde von diesem ausgezeichneten Gelehrten in vorliegender Mittheilung noch öfters zu reden haben, denn er ist der einzige Forscher, der für die Beurtheilung des Nahrungsbedürfnisses der Kinder jenseits des Säuglingsalters wissenschaftliche Grundlagen geliefert hat. Er fand, wenn er die abgemessenen Mengen von Kuhmilch, welche die Säuglinge nach der Entwöhnung tranken, auf ihren Kalorieenwerth berechnete und mit demjenigen der vorher genossenen Muttermilch verglich, dass die Zufuhr an Energie mit einem Male eine unverhältnissmässig viel grössere wurde. Trotzdem nahmen aber die Kinder nicht etwa der grösseren Zufuhr entsprechend mehr zu, sondern eher weniger. Diese hatte also nur zu erhöhter Wärmeerzeugung führen können. Verfolgte er aber die Zufuhr weiter bis zu dem Zeitpunkte, wo gemischtere Kost an Stelle der ausschliesslichen Kuhmilchernährung trat, so fand sich, dass dann die übermässige Zufuhr von Energie allmählich sich wieder ausglich, und etwa im Alter von 1½ Jahren auf das Verhältniss zurückkehrte, wie es während der natürlichen Ernährung bestanden hatte.

Man wird wahrscheinlich nicht fehl gehen, wenn man annimmt, dass im Allgemeinen bei der Entwöhnung analog verfahren wird, wie es in den von *Camerer* citirten Fällen ausgemessen war. Jedenfalls entsprachen sie im einen Falle den Gewohnheiten in Süddeutschland, im andern in Mitteldeutschland, in einem dritten am Rhein. Man kann Aehnliches auch aus anderen Angaben berechnen, z. B. aus den an interessantem Detail reichen Tabellen von *Carstens*[2] über die

[1] *Jahrbuch für Kinderheilkunde*, Band xxx, S. 369 und *Der Stoffwechsel der Kinder*. Tübingen, 1894.

[2] "Ueber Vortheile und Nachtheile der Ernährung mit sterilisirter Milch." *Verhandlungen der xv. Versammlung der Gesellschaft für Kinderheilkunde in Düsseldorf*. Wiesbaden, Bergmann, 1899.

Resultate einer Ernährung in grösserem Maasstabe mit Kuhmilch aus einem Leipziger Musterstalle. Er berichtet über 37 vorher an der Brust und sodann künstlich genährte (Tabelle II) Fälle, deren Zunahme bis zum Jahresende und darüber hinaus mit der Wage bestimmt wurde—Angaben, aus deren Würdigung man schliessen darf, dass es sich um physiologische, nicht pathologische Verhältnisse gehandelt hat. Die verabreichte Tagesmenge giebt der Autor auf 1 bis $1\frac{1}{4}$ Liter an. Alter und Gewicht der Kinder, Zusammensetzung der Nahrung ist genau bekannt, so dass sich für eine Anzahl von Fällen der auf den Quadratmeter Körperoberfläche zugeführte Kalorieenwerth der Nahrung bei der Entwöhnung berechnen lässt. Es stellt sich, wenn man die tägliche Quantität nur zu einem Liter annimmt (die aber nach der Angabe des Autors selbst öfters überschritten worden ist) in mehreren Fällen zwischen 1400 und 1500 Kalorieen (pro □ M.), in einem Falle schon in der 9. Woche (No. 3 der Tabelle), in einem zweiten in der 12. Woche (No. 20), in einem dritten in der 30. Woche des Lebens (No. 27). Also auch hier die gleichen Verhältnisse, wie sie *Camerer* beobachtete. Allerdings kamen bei *Carstens* auch Fälle vor, wo beim Entwöhnen die normale Energiemenge zugeführt wurde—unter der Annahme der Tagesmenge von 1 Liter—z. B. No. 18 seiner Tabelle 11, nämlich 1190 Kalorieen. Dieses Kind entwickelte sich aber gerade besonders gut und regelmässig bis zum Ende des 1. Lebensjahres.

Man muss aus diesem Ansteigen der Energiezufuhr um die Zeit der Entwöhnung den Schluss ziehen, dass hier die allgemein übliche Sitte nicht das Richtige getroffen hat, dass vielmehr bei dem Uebergang von der Brusternährung zur Kuhmilchernährung *zu grosse Nahrungsmengen* verabreicht werden. Die Versuchung hierzu ist offenbar in der Form der Darreichung gelegen. Während der Säugling an der Mutterbrust seine Saugmuskeln anstrengen muss, um seine Nahrung zu holen, ist das, wenn er sich einmal an die Flasche gewöhnt hat, nicht oder in viel geringerem Grade der Fall, er kann bequem in kürzerer Zeit wesentlich grössere Volumina in den Magen bekommen, als vorher und so bildet er sich selbst zum Vieltrinker aus. *Carstens* giebt deshalb den Rath, beim Uebergang zur künstlichen Ernährung den Säugling langsam, mit Absätzen trinken zu lassen, weil er so leichter zum Sättigungsgefühl komme.

Es dürfte aber nicht daran zu zweifeln sein, dass obige Ueberernährung die um die Zeit der Entwöhnung so häufig Platz greift, sehr leicht in eine Schädigung des Kindes umschlagen kann. Und wahrscheinlich werden die häufigen um diese Zeit sich einstellenden

Verdauungsstörungen zu einem erheblichen Theil durch diesen Fehler hervorgerufen. Der Laie erblickt selbstverständlich keinen Missgriff in solchem Verfahren, denn da ihm nicht bekannt war, wie viel sein Kind an der Brust Nahrung zu sich nahm, so denkt er gar nicht daran, dass die neue Ernährung eine zu reichliche sein kann. Vielmehr deutet er jedes Geschrei des Kindes als Hunger und macht den Fehler immer grösser.

Es ist Sache des Arztes, überall wo ihm die Gelegenheit sich bietet, die Entwöhnung nach dieser Richtung zu überwachen. Die Sache ist sehr einfach. Man kennt die Quantität Muttermilch, die der Säugling in den verschiedenen Monaten zu sich nimmt, man kennt den durchschnittlichen Nährwerth der Muttermilch, und kann daraus die Kalorieen berechnen (in dem citirten Buch von *Camerer* sind sie auf Seite 108 für die einzelnen Monate angegeben). Die Nahrung muss nun so eingerichtet werden, dass sie zur Zeit der Entwöhnung diese Werthe nicht überschreitet. Wem die Kalorieenberechnung zu wenig geläufig, der kann sich auch allenfalls damit helfen, dass er die Nahrung in toto den Betrag eines Liters oder eines Kilo nicht übersteigen lässt.

Wird die Entwöhnung allmählich vollzogen, so kann man die zur Brustnahrung nöthige Zugabe an anderer Nahrung nur dadurch genau feststellen, dass man durch die Wage (wägen vor und nach jedem Anlegen) die aus der allmählich versiechenden Brust in 24 Stunden abgegebene Nahrungsmenge feststellt, und soviel wenig verdünnte oder unverdünnte Kuhmilch zugiebt, dass der Gesammtbetrag ein Liter (oder Kilo) ausmacht. So geregelt wird die Entwöhnung in vielen Fällen sehr viel glatter sich gestalten.

Eine weitere Frage tritt gegen Ende des 1. Lebensjahres heran. Wann soll man zur gemischteren Nahrung übergehen, und die reine Milchernährung verlassen? Sie wird zur Zeit von verschiedenen Richtungen unter den Aerzten noch sehr abweichend beantwortet. Es giebt noch zahlreiche Stimmen, welche mindestens bis zum Ende des 1. Lebensjahres oder auch weit darüber hinaus von irgend etwas Anderem, als reiner Milch, nichts wissen wollen. Ich selbst habe schon seit Jahren dieser Diaetetik strikter Observanz nicht mehr gehuldigt. Aber zu ernsten und ausschlaggebenden Zweifeln an der Richtigkeit einer sehr lang fortgesetzten ausschliesslichen Milchernährung regten mich erst die Erfahrungen bei der Behandlung der *Barlow*'schen Krankheit, oder wie die Amerikaner sie bezeichnen, des infantilen Scorbutes, an. Den ersten Fall dieser Art sah ich im Jahre 1876, aber ohne damals zu wissen, womit ich es zu thun hatte. Diagnosticirt habe ich die Krankheit zum ersten Male im Jahre 1889. Und gleich bei diesem 1-jährigen Patienten fiel mir auf, in welch' glänzender Weise die Unterbrechung

der bis dahin geübten monotonen Milchdiät, durch die Zufuhr frischer Fruchtsäfte und Gemüse, sowie sie *Barlow* in der Keating'schen Encyclopædia empfohlen hatte, die rasche Heilung herbeiführte. Seitdem hat sich die gleiche Diaetetik in einer grossen Anzahl analoger Fälle ausnahmslos bewährt. Ich fing alsbald auch an, ähnliche diaetetische Vorschriften—mit Auswahl und Vorsicht manchmal schon vom 7. 8. Monate an—bei Rhachitis, Anaemie, sonstigen allgemeinen Ernährungsstörungen anzuordnen. Auch hier erwies sich der Erfolg meist sehr befriedigend. Später kam zur weiteren Stütze für die Verwerfung einer übertrieben langen Fortsetzung ausschliesslicher Milchernährung der Hinweis *Bunge's* auf den sehr niedrigen Eisengehalt der Milch. Mit ihm schien die Erfahrung übereinzustimmen, dass Kinder, die in der Mitte ihres ersten Lebensjahres ein gutes Inkarnat dargeboten hatten, oft bei einseitiger Milchdiaet um den Beginn des zweiten Lebensjahres ein sehr blasses und welkes Aussehen bekamen.

So ist es nach meiner Auffassung räthlich, schon vom 3. Vierteljahr des Lebens an bei Kindern die nicht recht vorwärts kommen wollen, Anfänge der Rhachitis oder auch nur auffällige Hautblässe erkennen lassen, kleine Abwechselungen in der einförmigen Lebensweise eintreten zu lassen, sei es auch nur durch einige Theelöffel Fruchtsaft, Gemüsebrei, einen aufgeweichten Zwieback oder dergl.

Wann consistentere Zusätze zum Speisezettel erlaubt werden dürfen, das richtet sich wohl in der Hauptsache in sehr natürlicher Weise nach dem Verhalten der Zahnentwicklung. Deshalb wird im Allgemeinen bei normaler Entwicklung das Ende des ersten Lebensjahres oder der Anfang des zweiten herankommen, bevor man neben Milch, Suppe und Brei feingewiegtes Fleisch, kleine Stückchen Semmel, Kakes oder Brod zur weiteren Abwechslung gestattet.

Doch kann es in pathologischen Fällen unter Umständen geboten sein, sich von der Rücksicht auf die Ausrüstung der Mundhöhle mit den eben genannten Verordnungen zu emancipiren.

Mit dem zweiten Lebensjahre kommt allmählich die Zeit heran, wo das Kind mehr an den Tisch der Erwachsenen herangezogen werden kann, und mit dem dritten Lebensjahre wird es in der Mehrzahl der Fälle an den Mahlzeiten der Familie mit gewissen Beschränkungen theilnehmen.

Es fragt sich nun, wie ist von da an bis in die spätere Kindheit und bis zum Uebergang in das erwachsene Alter die Ernährung am zweckmässigsten einzurichten. Haben wir bestimmte Anhaltspunkte für unsere Verordnungen, können wir uns wenigstens nach einer grösseren Zahl von gut beobachteten und in ihren Resultaten bekannten Einzelfällen richten, oder sind wir lediglich auf unsere allgemeinen von

der Ernährungslehre der Erwachsenen entnommenen Informationen angewiesen?

Man muss sagen, dass es schon mit den Berichten über die einfachsten Thatsachen, mit Messungen und Wägungen der Nahrungsmengen, die von einzelnen Kindern in den verschiedenen Altersstufen aufgenommen sind, und einen Schluss auf das Nahrungsbedürfniss zulassen würden, sehr dürftig aussieht. Es würden nicht viel mehr als etwa zwei Dutzend von concreten Fällen heraus kommen, wo von einzelnen Beobachtern wenigstens während einer Reihe von Tagen derartige Messungen bei gesunden Kindern angestellt und veröffentlicht worden sind. Die bekanntesten sind die Mittheilungen von Anna *Schabanowa*, Sophie *Hasse*, *Uffelmann*. Neuerdings hat einer meiner Schüler, *Herbst*, [1] eine Reihe sorgfältiger Messungen an fünf Kindern ausgeführt.

Bei der Spärlichkeit dieser Unterlagen ist es aber mit um so grösserem Danke zu begrüssen, dass wir wenigstens eine klassische Arbeit über die Ernährung des Kindes in den fraglichen Altersstufen besitzen, die von dem schon genannten W. *Camerer* herrührt, und die man als dessen Lebenswerk bezeichnen kann. Die Bedeutung dieser Leistung wird meines Erachtens von den praktischen Paediatern—auch von denen, die sie citiren—noch nicht genügend gewürdigt.

Camerer hat an seinen eigenen fünf Kindern im Verlaufe von zwei Jahrzehnten methodische Stoffwechseluntersuchungen angestellt, die sowohl Einnahmen wie Ausgaben quantitativ ermittelten, sich jedes Mal über grössere Perioden erstreckten, und alle Altersstufen in ihr Bereich zogen. Im Ganzen gebietet er über 840 einzelne Versuchstage. Die Kinder waren theilweise etwas zart, aber gesund und haben sich in regelrechter Weise bis in das erwachsene Alter, augenblicklich zum Theil in das dritte Decennium hinein entwickelt. Es handelt sich also um eine normal erfolgreiche Ernährung, allerdings, wie C. selbst anführt, bei sparsamem Haushalt, und die gewonnenen Mittelzahlen dürfen vielleicht mehr nach der untern, als nach der oberen Grenze hin gerichtet angesehen werden. Aber sie dürfen vorderhand als Standardzahlen betrachtet werden. Es wird nicht so bald eine ähnliche Untersuchung mit gleicher Ausdauer, Consequenz, und Wissenschaftlichkeit wieder angestellt werden.

Die Resultate sind in zahlreichen Abhandlungen in der *Zeitschrift für Biologie* veröffentlicht und in dem obenerwähnten Stoffwechselbuche zusammengestellt. Sie bilden eine Fundgrube für die verschiedensten Fragen, die mit der Physiologie der Ernährung und Entwickelung der Kinder zusammen hängen.

[1] *Jahrbuch f. Kinderheilkunde*, Band xlvi, S. 245.

Für den in dieser Abhandlung ins Auge gefassten Zweck finden wir nun eine Reihe fester Anhaltspunkte in den von *Camerer* vorgelegten Resultaten. Sie bestehen in genauen zahlenmässigen Angaben über das Nahrungsbedürfniss, und zwar an allen drei Hauptnährstoffen, in allen Altersstufen vom 2.–14. Lebensjahre. Ich habe die von *Camerer* in einer seiner Haupttabellen mitgetheilten Zahlen so umgerechnet, dass sie *auf das Kilo Körpergewicht* in den einzelnen Altersstufen anwendbar sind. Aus der so gewonnenen Tabelle kann man für sehr verschieden kräftige Kinder des betreffenden Alters das Nahrungsbedürfniss berechnen und damit den Umstand, dass es bei C. um etwas zarte Kinder sich gehandelt hat, bis zu einem gewissen Grade eliminieren. Ich habe bei der Berechnung die von *Camerer* für Knaben und Mädchen getrennt angegebenen Zahlen zusammengezogen. Wem genaueres Detail erwünscht ist, der kann sich die Zahlen, für beide Geschlechter getrennt, leicht aus dem Buche selbst berechnen.

Tabelle.—Ein Kilo Kind braucht an Nahrung

im Lebensjahr	Eiweiss.	Fett.	Kohlenhydrate.	Kalorieen.
	Gramm.	Gramm.	Gramm.	
2– 4	3.6	3.1	9.2	75.3
5– 7	3.2	2.2	10.8	73.
8–10	2.7	1.3	10.2	60.
11–14	2.5	1.0	8.0	55.

Auffällig ist in dieser Uebersicht der verhältnissmässig hohe Eiweissconsum der C.'schen Kinder. Er übersteigt denjenigen des Säuglings an der Mutterbrust, aber auch den des Erwachsenen nicht unerheblich. Trotzdem wird sich zeigen, dass, auf eine concrete Mahlzeit umgerechnet, dieser Betrag immer noch geringer ist, als ihn viele Kinder, aus den bemittelten Ständen wenigstens, täglich zu sich nehmen oder auch von ihren Aerzten verordnet bekommen. Niedrig ist der Antheil der Fette an der Gesammtnahrung. Die höheren Werthe bis zum 7. Lebensjahre beruhen wohl vorwiegend darauf, dass die Milch noch einen grossen Bruchtheil der Gesammtnahrung ausmachte. Vom 8. Jahre an wird der Fettbetrag der Nahrung auffällig niedrig. Dafür war der Consum an Kohlenhydraten während der ganzen Kindheit ein hoher.

Das gegenseitige Verhältniss zwischen Fett und Kohlenhydraten

wird, auch wenn man sich im Allgemeinen an die Tabelle als Norm hält, ohne Bedenken erheblich geändert werden können. Besonders in Bezug auf dieses werden je nach Landessitte und Aufenthalt andere Speiseformen gewählt werden, als in den mehr ländlichen Verhältnissen einer kleineren würtembergischen Stadt, wo *Camerer* seine unermüdlichen Beobachtungen anstellte. In der That unterscheiden sich auch die von anderen Autoren berichteten Kostformen gesunder Kinder nicht unerheblich von den hier aufgestellten Normen.

Sehr nahe kommt den *Camerer*'schen Werthen *Uffelmann*,[1] der auch seine eigenen Kinder zur Messung der Nahrung beobachtet hat. Er berechnet für

ein 8-jähriges Kind (mittl. Gewicht 22.5 Kilo) per Kilo 2.6 Eiweiss, 1.9 Fett, 6.6 Kohlenhydrat.
ein 12-jähriges Kind " " 33 " " 2.2 " 1.4 " 7.4 Kohlenhydrat.
Er gab also schon etwas mehr Fett, aber weniger Kohlenhydrat.

Dagegen fand *Sophie Hasse*[2] viel höhere Werthe, freilich auch ungewöhnlich hohe Gewichte. Auf's Kilo kommen bei dieser Autorin

Bei	$2\frac{1}{2}$-jährigen Kindern (2)	3.7 Eiweiss,	2.9 Fett,	11.5 Kohlenhydrat.	
"	$3\frac{1}{2}$-jährigem Kind	2.9 "	2.1 "	11.8	"
"	4 " "	3.8 "	3.4 "	10.2	"
"	8 " "	2.6 "	2.7 "	7.0	"
"	$10\frac{1}{2}$ " "	2.2 "	2.7 "	6.9	"

Hier war also die Nahrung durchschnittlich reichlicher, als bei *Camerer*, bei den jüngeren Kindern an Kohlenhydraten, bei den älteren ganz besonders an Fett, während im Gegentheil die Kohlenhydrate zurück traten. Das Gewicht dieser Kinder stand aber auch weit über dem Durchschnittsgewicht der betreffenden Altersklassen.

Herbst (*l. c.*) berechnet

Für 2–4-jährige Kinder (2)	pro Kilo	3.8 Eiweiss,	4.0 Fett,	9.1 Kohlenhydrat.	
" 10-jährigen Knaben	"	2.2 "	2.5 "	8.2	"
" $10\frac{3}{4}$-jähriges Mädchen	"	1.4 "	1.6 "	5.7	"
" $12\frac{1}{2}$ " "	"	1.5 "	1.8 "	4.4	"
" $14\frac{1}{2}$ " "	"	1.4 "	1.4 "	4.5	"

Auch hier finden wir also durchweg einen höheren täglichen Consum von Fett, als bei *Camerer*. Dagegen ist bei den älteren Mädchen der Bedarf an Kohlenhydrat auffällig niedrig und wird im Verhältniss zu *Camerer* nicht durch den reichlicheren Fettgenuss gedeckt.

[1] Munk und Uffelmann. *Die Ernährung*, S. 318, 1887.
[2] *Zeitschrift f. Biologie*, Bd. xviii.

Uebrigens sind die Angaben aller dieser Autoren insofern nicht mit den grundlegenden Zahlen *Camerers* vergleichbar, als die Untersuchung sich über viel kürzere Zeiträume erstreckte, als bei den lange Perioden im Verlaufe vieler Jahre umfassenden *Camerer*'schen Messungen. Es wird vor der Hand wohl das Richtige sein, bei den Ausmaassen der Kost für gesunde Kinder sich—unter Berücksichtigung der Gewichte der jeweilig in Betracht kommenden Fälle—an den *Camerer*'schen Canon zu halten.

Vergleicht man aber damit z. B. die Kostverordnung die *Wilh. Steffen*[1] für das erste Kindesalter giebt, unter Zugrundelegen der mittleren Körpergewichte der jedesmaligen Altersklasse, so lässt dieser Autor täglich pro Kilo geniessen:

1-jährige	Kinder	6.6	Eiweiss,	6.0	Fett,	5.9	Kohlenhydrat.	
2	"	"	5.6	"	5.5	"	6.5	"
3	"	"	5.6	"	5.3	"	8.8	"
4–7	"	"	5.3	"	5.1	"	11.0	"

Das sind, an unserem Canon gemessen, ganz exorbitante Mengen von Eiweiss und Fett, und nimmt man noch die grossen Kohlenhydratmengen im 4–7. Jahre hinzu, so bedient sich dieser Autor bei seiner Diätetik zweifellos einer fortdauernden starken Ueberernährung. Trotzdem empfiehlt er sie auf Grund einer 13-jährigen Erfahrung; und ausserdem, gestützt auf die Autorität seines Vaters, des hervorragenden Paediaters. Um diese Gegensätze zu erklären, darf man vielleicht in etwas die verschiedene Lage der Wohnorte in Rücksicht ziehen, wo einerseits *Steffen* Vater und Sohn, andererseits *Camerer* thätig sind. Aber sie reichen nicht völlig dazu aus. Sie beruhen vielmehr, soweit *Steffen* in Betracht kommt, auf traditionell gewordene Grundanschauungen denen ich auch in Berlin hier äusserst häufig begegne. Man pflegt auch hier sehr vielfach die Kinder in der von *Steffen* gerühmten Weise zu ernähren. Aber schon sehr oft habe ich in solchen Fällen Gelegenheit gehabt, Appetitlosigkeit, Verstopfung, Nervosität, Schlaflosigkeit und Blutarmuth durch eine totale Umänderung der bisherigen überreichen Eiweiss- und Fettdiät im Sinne von *Camerer* in kurzer Zeit zu beseitigen. Schon vor einer Reihe von Jahren hat ein Berliner Physiolog, *Zuntz*,[2] die praktischen Aerzte in sehr eindringlicher Weise vor der Ueberernährung gerade im ersten

[1] *Jahrbuch f. Kinderheilkunde*, Bd. xlvi, S. 342–3. Dieser talentvolle junge Arzt ist seit der Niederschrift vorliegender Zeilen zum tiefen Schmerz seines allen Lesern dieser Zeitschrift bekannten Vaters einer akuten Erkrankung erlegen.

[2] Zuntz, "Welche Mittel stehen uns zur Hebung der Ernährung zu Gebote?" Vortrag im Verein für innere Medicin in Berlin. *Deutsche medic. Wochenschrift*, Nov. 20, 1893.

Kindesalter gewarnt; und ich kann mich auf Grund meiner klinischen Erfahrung diesem physiologischen Urtheil nur durchaus anschliessen. Neuestens spricht sich *Czeray*[1] in ähnlichem Sinne aus. Man erinnere sich nur der grossen und im Effekt ganz unnöthigen Arbeit, die das Eiweiss nach den Berechnungen *Camerers*[2] und den schlagenden Versuchen *Pawlows*[3] zu seiner Verdauung beansprucht, seines verhältnissmässig niedrigen kalorischen Werthes und der geringen Mengen, die für den Ansatz beim Wachsthum nöthig sind, um bei der Ernährung der Kinder jenseits des Säuglingsalters ebenso, wie während desselben, eine übermässige und ganz überflüssige Zufuhr von Eiweiss in gleicher Weise zu vermeiden. Aehnliches gilt auch für das Fett, gegen dessen Bewältigung bei übergrosser Zufuhr der Darm sich einfach dadurch auflehnt, dass er es in Masse wieder unbenutzt abgehen lässt.

Im Gegensatze zu den bei *Steffen* zu findenden und von vielen praktischen Aerzten, wenigstens Norddeutschlands, gebrauchten Kostverordnungen habe ich versucht, zwei Beispiele zusammen zu stellen, die zeigen sollen, wie die Kostverordnung nach den von *Camerer* gefundenen Regeln bei einem 2-jährigen und bei einem 7-jährigen Kinde von mittlerem Gewichte etwa zu lauten hätte.

Für das 2-jährige: 1 Liter Milch, 50 Gramm Weissbrod oder Schwarzbrod, 2 Zwiebäcke, 20 Gramm Schinken oder ein Ei, 10 Gramm Butter, 100 Gramm Griessuppe, 20 Gramm Kartoffelmuss, und 20 Gramm Apfelmuss mit 10 Gramm Zucker.

Für das 7-jährige: 600 Gramm Milch, 120 Gramm Weissbrod, 100 Gramm Schwarzbrod, 30 Gramm Butter, 20 Gramm Zucker, 200 Gramm Reissuppe, 70 Gramm Kalbsbraten, 100 Gramm Kartoffeln, 200 Gramm Aepfel.

Und nun vergleiche man damit die Nahrungsmengen, die Kinder dieses Alters in den Häusern der auch nur mässig Bemittelten und gar der Wohlhabenden aufnehmen. Mässige Ueberschreitungen werden ja nicht schädlich sein, aber wie oft wird man sie unverhältnissmässig finden. *Zuntz* sagt mit Recht, es wäre vielleicht zu empfehlen bei der Ernährung der Kinder lieber die Gebräuche der Minderbemittelten nachzuahmen, als die der Wohlhabenden und Reichen!

Nicht allerdings gelten diese Regeln für die Kranken und besonders die reconvalescenten Kinder. Vergleicht man die von *Baginsky* und *Dronke*[4] mitgetheilten Kostmaasse, besonders bei den letzteren, so

[1] *Jahrb. f. Kinderheilkunde*, Bd. li, 1. Heft.
[2] *Jahrbuch f. Kinderheilkunde*, Bd. xxx u. Bd. l.
[3] *Die Arbeit der Verdauungsdrüsen*, Wiesbaden, 1898.
[4] *Archiv f. Kinderheilkunde*, Bd. xvi u. Bd. xxiv.

sieht man, dass Zunahme nur dort erfolgt, wo über die obigen Zahlen hinausgehende, denen *Steffens* ungefähr analoge Nahrungsmengen gereicht wurden. Doch kann hierauf an dieser Stelle nicht weiter eingegangen werden.

Für die Gesunden aber möchte ich—mindestens in Bezug auf die stickstoffhaltigen Nährstoffe—zum Wahlspruch erheben: "lieber etwas knapp, als zu reichlich," um aus unseren Kindern ein gesundes Geschlecht erwachsen, um aus der Decadence *fin de siècle* eine neue Blüthe im aufgehenden Jahrhundert emporstreben zu sehen.

CLINICAL OBSERVATIONS UPON THE OPERATIVE TREATMENT OF TUBERCULOUS PERITONITIS.

By AUGUSTUS CAILLÉ, M.D.,

Professor of Pediatrics, N. Y. Post-Graduate Medical School and Hospital.

BY reason of its peculiar clinical behavior, tuberculous peritonitis claims in a high degree the interest of the physician and surgeon, inasmuch as the brief exposure to the air or sunlight of a portion of the accessible infected area is apparently the starting-point of a reparative or healing process in cases that have resisted other therapeutic efforts.

The opening of the abdomen is, as a rule, followed by an arrest of local disease symptoms, and may be followed by a disappearance of the tuberculous deposits on the peritoneum, as shown by certain cases in which the abdomen has been opened for some reason or other for the *second time.*[1]

Two points of special interest present themselves to the clinician;

1. Behavior of tuberculous peritonitis cases before the opening of the abdomen, or, in other words, the *diagnostic features* of the disease.

2. Its clinical behavior after laparotomy.

This report embraces not all the cases observed by the writer, but only such as were admitted to the writer's service at the Babies' Wards, Post-Graduate Hospital, and carefully studied before and after operation, and which have subsequently been under my observation for a period of from one to three years.

For the sake of brevity and to avoid repetition I will state that every case underwent a careful clinical examination, including the examination of urine, blood, fæces, and puncture fluids, *and no special mention will be made in the brief histories whenever the co-existing conditions were found to be normal.* Much of the laboratory work was done in the laboratory of the N. Y. Post-Graduate School, by Dr. H. T. Brooks.

[1] "Tubercular Peritonitis Cured by Laparotomy." Dr. F. Jameson. *Buffalo Med. Journal*, May, 1899.

Case 1. Maurice J., six years old. Admitted February 9, 1897. Sick for the past five months. Gradual swelling of abdomen, loss of flesh, no pain.

Status on Admission:

Weight, 41 lbs.

Temperature, 100°, with subsequent rises to 103°.

Pulse, 100.

Respiration, normal.

Physical examination showed normal conditions except as follows: The abdomen was swollen and somewhat tense, not painful on percussion or on palpation, and contained fluid. On auscultation much peristaltic unrest was noticeable. Double inguinal hernia also existed. A diagnosis of tuberculous peritonitis was made by exclusion, and between February 9th and April 16th creosote, creosote carbonate, guaiacol, ichthyol, and arsenic were exhibited internally and externally and by means of rectal irrigations, but without apparent benefit. On the contrary, a hard mass appeared soon after admission, in the scrotum, close to the testicles on either side, which slowly spread and was looked upon as a *tuberculosis of hernial sac.*

On May 16th the operation for double inguinal hernia was performed by Dr. B. F. Curtis by the Bassini method. The peritoneum forming the hernial sacs on both sides and in the abdomen as far as the finger could reach when introduced into the inguinal rings, presented the usual pathological appearance of *tuberculous peritonitis.* The membrane was unusually thickened, covered with nodules and deposits of fibrin, and congested. There was some clear serum in the abdominal cavity; the testicles were healthy. The hernia was not congenital on either side. The wounds healed in due time. I saw patient two years after operation and found him to all appearances perfectly well. Physical examination revealed no abnormality. *It is to be noted that in this case the general abdominal cavity was not encroached upon at the time of operation.*

Case 2. Franz B., two and a half years old. Admitted April 19, 1898.

Weight, 25 pounds.

Temperature, 99°, with occasional elevation.

Pulse, 112.

Respiration, 36.

Patient eats well and looks well.

A careful clinical examination was made, which was negative except as follows.

Abdomen symmetrically distended, no fluctuation. *A firm tumor* within the abdomen, on each side, and a third *tumor* in median line higher up; each tumor has well defined, sharp margins; operated upon April 27, 1898, by Dr. B. F. Curtis; no previous medication. The tumors were found to be large tuberculous deposits in the omentum, firm and vascular. Intestines studded with tubercles of all sizes. No fluid in abdominal cavity. Tumors not adherent to any organ and no enlargement of mesenteric glands. One independent tuberculous nodule was found high up in the omentum. Ten grammes of a 10 % glycerine-iodoform emulsion was put into the abdomen and the same was closed. Convalescence was uneventful. Eighteen months after operation the tuberculous tumors can still be felt but are *very much smaller.*

There is no other evidence of tuberculous disease.

Case 3. Isidor B., two and a half years old. Admitted February 29, 1892; on admission looked anæmic but not jaundiced; said to have had large stomach for one year.

Weight, 26 pounds.

Temperature, 102.4°. Temperature curve shows irregular low fever.

Pulse, 140.

Respiration, 48.

Complete clinical examination negative excepting abdomen, which was enormously distended; two quarts sero-sanguineous fluid were removed by trocar. The child has lost weight and strength and has pain. Constipation is noticed but no vomiting.

Operated July 19th, by Dr. Coley. Showed multiple encysted pus cavities and detritus and characteristic miliary tubercles; the wound was drained; a subsequent counter opening was made by Dr. Lloyd. After a lingering illness the child died. The operation was not followed by any improvement.

Case 4. Declon B., four years, admitted Aug. 8, 1898. Abdomen began to swell six months before admission, otherwise feels well. Clinical examination negative, excepting albumin and bloody epithelial casts in urine. Abdomen 24⅛ inches in circumference, distended with fluid; area of liver dullness extends from nipple to one inch below ribs.

Weight, 23 pounds.

Temperature, 98° (subsequent irregular low fever).

Pulse, 124.

Respiration, 38.

Operated Nov. 5, 1898, by Dr. Lloyd. Showed a characteristic tuberculous peritonitis. The abdomen was flushed with warm normal salt solution and closed. Uneventful recovery. The boy is in very good health at the present time.

Case 5. Elisabeth N., five and a half years old, admitted March 31, 1898. Her abdomen became large and tense a few months before admission to Babies' Wards. No pain.

Weight, 38 pounds.

Temperature, 99° (irregular low fever).

Pulse, 120.

Respiration, 24.

Careful clinical examination negative, except as follows:

Encysted fluid in abdomen extending no higher than umbilicus; line of percussion flatness not changed by putting patient in Trendelenburg or other positions. Dark brown fluid removed by puncture. Gonococci in vaginal discharge.

Operated April 27, 1898, by Dr. B. F. Curtis. Peritoneum thick and adherent, large amount of dark brown serous fluid evacuated. Adhesions broken by finger. Intestines covered with miliary tubercles. Abdominal cavity irrigated with saline solution and dried out, 10% emulsion of iodoform and glycerine; abdominal cavity closed. The fluid evacuated had its source in a large cavity, which was separated from the general cavity above by adhesions which were not ruptured. A sinus from the abdominal wound persisted for about one year and closed spontaneously the middle of April, 1899. Directly after closure a cough set in, and on examination May 11, 1899, moist râles were heard over entire right lung. The sinus opened again and discharged; it was subsequently curetted by Dr. Wilson, and has closed completely. At the present she is well and has gained in weight.

Case 6. Julia D., three years old, admitted April 24, 1899.

20½ pounds' weight on admission.

Temperature, 98°. (Fever curve irregular, from normal to 103° F.)

For two months before admission she complained of pain in abdomen, vomiting, and constipation. The clinical examination was negative, excepting abdomen, which was tender on palpation. Under ether the intestinal convolutions could be felt, also bands of tissue which proved to be adhesions.

The abdomen was opened April 24th by Dr. Dunham and a tuberculous peritonitis with intestinal adhesions found. The wound healed, leaving a fæcal fistula with adhesions. Up to the present time her general condition is splendid. The fistula persists and will require further operative interference.

Case 7. Joseph F., six and a half years old. Admitted June 15th. The boy's mother died of pulmonary tuberculosis. For six months past he has had spasmodic pain about the umbilicus. He is constipated, pale, and has no appetite.

A careful examination reveals nothing of note, excepting a tender abdomen.

Weight, 23 pounds.

Temperature, 100.4°. (Irregular low fever curve.)

Pulse, 100.

Respiration, 24.

The appendix can be felt, *but is not as tender as other parts of abdomen.* The percussion sound is that found over collapsed intestine.

Operation was performed July 5th, by Dr. Wilson, and an adhesive miliary tuberculosis of peritoneum was found.

The abdominal cavity was flushed with saline solution and closed. The boy is quite well at present writing.

Case 8. Joseph C., two and a half years old, admitted May 17, 1898. Three weeks before admission the abdomen began to enlarge and child was feverish. The scrotum began to fill when child cried; the hernia now remains down continually. Skin muddy-looking.

Weight, 24 pounds.

Temperature, 101⅕°. (Irregular fever curve.)

Pulse, 40.

Respiration, 36.

Abdomen distended, flatness on percussion, free fluid in peritoneal cavity. Reducible right inguinal hernia.

Hæmoglobin, 60 %.

Red corpuscles, 5,417,000.

White corpuscles, 6,250.

Urine, trace of albumin, hyaline casts.

Operation May 27, 1898, Dr. B. F. Curtis.

Finger detected a large cyst cavity, extending to umbilicus, made up of small cysts containing considerable fluid. Fluid evacuated; 10 % emulsion of glycerine-iodoform introduced. The thickened peritoneum was closed with continuous catgut suture, skin also. A nodule from omentum proved to be tuberculous on microscopic examination. Hernia not operated upon.

Examined May 11, 1899. Musical rhonchi on deep inspiration over scapula on both sides. Child pale but improved. Thickening of tissues in line of wound and line of old sinus. At present writing patient is pale and anæmic, otherwise in apparent health.

Case 9. Isabella C., nine years, admitted Jan. 24, 1898.

Weight, 24 pounds.

Temperature, 97° to 103°. (Irregular fever curve.)

Pulse, 90–120.

Respiration, 30.

For past six months has had severe pain in abdomen and four to five stools during day and as many at night, losing flesh rapidly. Abdominal tenderness marked. Abdomen flabby; palpation reveals nothing noteworthy. No amœba or tubercle bacilli in stools. No plasmodia in blood. Red cells, 5,025,000. A careful examination showed no other abnormality. As the parents of the girl refused to permit an operation for the suspected tuberculous condition, all known methods of internal, percutaneous, and rectal medication were persisted in for the greater part of two years.

In November, 1898, the girl looked very anæmic and the abdomen was tender and tense, but her weight was 36 pounds. In March, 1899, her weight was down to 28 pounds. She had much diarrhœa and vomiting and intense paroxysmal pain, also some cough and general anasarca. In February, 1899, the parents of the patient gave permission to open the abdomen (Dr. Wilson). The intestines were found to be matted together by tuberculous tissue and the adhesions were found to be too tense to be broken up. There was no pus. The accessible portion of the abdominal cavity was flushed with saline solution and the abdomen was closed. The girl died in October, 1899, from exhaustion. No autopsy.

Case 10. Girl of nine, Flora B., who was afflicted with a not very extensive tuberculous

infection of right lung apex and whose abdominal symptoms came on gradually. She had pain in paroxysms, pain on pressure. Various dull areas next to tympanic spots in abdomen. Occasional fever and loose bowels. On abdominal section the intestines were found matted together, the interspaces were filled with a dark yellow fluid and some detritus. Microscope revealed tubercle bacilli. Iodoform-glycerine introduced; drainage; marked improvement, but died from general tuberculosis two years later.

The following three cases have also been under observation for at least one year, but I have since lost track of them and am unable to report as to their present condition, and whether they are alive or dead.

Case 11. Harry V., five years old, admitted October 12, 1897. Patient was apparently well up to two months before his admission to the Babies' Ward, when he lost his appetite and began to vomit. He coughed and expectorated. Had fever and sweating and dyspnœa.

Weight, 40 pounds.

Temperature, 100° (irregular fever curve).

Pulse, 140.

Respiration, 40.

A careful clinical examination revealed: abdomen distended, superficial veins prominent, fluid in abdomen. Area of fluid dulness changes with position.

Sonorous râles and rhonchi are heard all over the chest. He looks anæmic and poorly nourished. Abdominal section by Dr. Curtis showed tuberculous peritonitis. Iodoform and glycerine introduced. Closure of abdomen. Perfect healing of wound. Discharged improved and lost sight of a year ago.

Case 12. Hattie V., four and a half years old, admitted March 2, 1898.

Weight, 28 pounds.

Temperature, 100° (irregular fever curve).

Pulse, 132.

Respiration, 32.

This patient had a tuberculous look or habitus, and it was stated that her abdomen had begun to swell some time before admission to the hospital. She was constipated, had fever, but no cough, and her abdomen was found distended with fluid and gas. Albuminuria was also noticed.

Operation by Dr. Curtis March 17, 1898. The peritoneum was found thickened, the bowels agglutinated, and much fluid was evacuated by breaking down adhesions. Miliary tubercles were seen in great numbers on loops of intestine, and tuberculous elements were detected by Dr. Brooks, Post-Graduate Laboratory.

Iodoform-glycerine was put into abdomen and the latter sutured. Healing by primary union. General condition much improved after operation. Discharged in good condition and lost sight of a year ago.

Case 13. Frank O., three years old, admitted August 23, 1898.

Weight, 23 pounds.

Temperature, 100.2° (irregular fever).

Pulse, 120.

Respiration, 28.

The child was sick, three months before admission into the hospital, with cough, distended abdomen, and alternating constipation and diarrhœa. Bilateral broncho-pneumonia; abdomen painful on palpation—contains fluid.

Operated by Dr. Lloyd. Discharged (unimproved); operation wound healed. General tuberculous infection. Case lost sight of.

Résumé.—The diagnosis of tuberculous peritonitis is based upon the

abdominal symptoms, such as distension, pain, and disturbed bowel action, presence of fluid and loss of weight, *and is made by exclusion*, except in those cases in which the tubercle bacilli are found and then the diagnosis is positive. *A febrile rise of temperature of an irregular type was found in all cases under careful observation.* There is nothing characteristic about the temperature curve.

Cases of chronic *non-tuberculous* serous peritonitis present usually the features of an ordinary ascites, the abdominal fluid being free, whereas it is usually not free in the tuberculous variety. It is rare to find the tubercle bacilli by microscopic examination of puncture fluid. In doubtful cases the opening of the abdomen is indicated and will do no harm. *Paroxysmal pain in the abdomen in children, in the absence of chronic appendicitis or abdominal fluid, is not indicative of tubercular disease and is frequently overcome by dieting and attention to and irrigation of bowels.* (Worms, intestinal indigestion, membranous enteritis, etc.)

The tuberculin test was employed in Cases 1 and 5; in the former with positive and in the latter case with negative result. The writer is unwilling to make a routine test with tuberculin in human beings in the present unsatisfactory state of our knowledge of its action.

To the three varieties of tuberculous peritonitis hitherto formulated by various observers:

1. Chronic tuberculous ascites (miliary form);
2. Fibro-caseous tuberculous peritonitis;
3. Fibro-adhesive tuberculous peritonitis;

must be classed a fourth variety,

4. Tuberculous peritoneal tumors.

Two such tumor cases have been seen by the author. Israel [1] reports one and there may be others on record which have not come to the writer's notice.

The infection of the peritoneum can come about by way of the circulation or from the gastro-enteric or genito-urinary tract. Only one of the children here reported upon had a tuberculous parent (mother), Case 7, and it is not too far-fetched to assume that milk and meat of tuberculous animals are a frequent source of infection.

The cases here reported show the futility of medicinal treatment, (most cases having been so treated before operation); and where some form of medication is followed by improvement or cure, one must not forget that spontaneous cures have also been reported and observed in

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[1] "Geschwulstartige Tuberculose des Colon ascendens." *Deutsche med. Woch.*, No. 1 1896.

cases presenting all the chemical evidences of the disease. Tuberculin and serum preparations were not exhibited as therapeutic agents.

Finally, the indication is *early* operation, which is no doubt of very great benefit to the patient when the tuberculous process is limited to the peritoneum. As regards the establishment of a *complete cure*, I am somewhat sceptical, because of the persistence of mild abdominal symptoms, of irritative catarrh or inflammation in bronchi, lungs, and pleura, and intestines, in a number of cases which remained under my observation two years after operation. If at the time of operation we have co-existing tuberculosis of the lung or pleura, the ultimate results are unsatisfactory, although some improvement usually takes place for the time being.

NOTES ON SOME EXPERIMENTS CONCERNING CELL EMIGRATION.

By I. ADLER, M.D., New York.

THE primary object of these experiments was to investigate if by the employment of different chemical substances the local emigration of the different forms of leukocytes can be influenced; if, in other words, one substance would exert a special chemotactic attraction upon lymphocytes, another upon polynuclear cells, etc. The great bulk of work done upon chemotaxis has for its object the study of the effect of various substances, introduced into the body in one way or another, upon the leukocytes in the circulating blood. In numerous experiments undertaken for the investigation of other problems, as, for instance, the different forms of inflammation, the healing of wounds in various tissues and organs, the fate of foreign bodies introduced into various tissues under varying conditions, etc., the different forms of leukocytes and their occurrence at various times and under varying influences have received most careful and painstaking attention. Borissow,[1] however, seems, as far as I can see, to have been the only one who has done work specially directed to the object stated above. The question is one of some interest inasmuch as investigations of recent years have brought to light a number of facts which seem to indicate that the different forms of leukocytes differ in their reaction upon varying influences. As such may be instanced the occurrence of eosinophile leukocytes in bronchial asthma, the behavior of plasma cells, of whose derivation from lymphocytes there seems little doubt, in inflammatory and reparative processes,[2] and the varying

[1] "Ueber die chemotaktische Wirkung verschiedener Substanzen auf amoeboide Zellen und ihren Einfluss auf die Zusammensetzung des entzuendlichen Exudats." *Ziegler's Beitraege*, Bd. xvi., 1894, p. 43 ff.

[2] Von Marschalkó: "Zur Plasmazellenfrage," *Centralblatt fuer allgem. Pathol. u. pathol. Anat.*, Bd. x., Nos. 21 and 22, where also literature on plasma cells is given. Councilman: "Interstitial Nephritis," *Transactions of Assn. of Amer. Phys.*, 1898. By the same author, "Character of Cellular Exudation in Acute Keratitis of the Rabbit," *Journal of the Boston Med. Soc.*, vol. iii., No. 5, 1899.

predominance of certain forms of leukocytes in the circulating blood as occurring in the course of different diseases.

On these and similar considerations it was thought proper to study the question once more, employing somewhat different methods. Borissow had used minute glass tubes, closed at one end, open at the other. These were partially filled with the substance whose chemotactic qualities were to be tested and the supervening space in the tubules filled with physiological saline solution. By means of a trocar the tubes were placed under the skin of dogs, rabbits, and frogs, in some of the latter animals also in the peritoneal cavity, always with strict attention to asepsis. At the close of the experiment the tubes were recovered, broken, or cut, and their contents spread out upon cover slips, dried, fixed, and stained according to various methods. He records a total of 111 experiments, which include a very great number of the most widely different substances. The main result attained by Borissow with reference to the question which interests us here is, that in his experiments no substance was found which exercises a special chemotactic attraction for any one species of cell. The interesting results as regards varieties in cell emigration in different species and different ages of animals can not be entered into here. The experiments briefly to be reported here were done exclusively on rabbits. Instead of glass tubules, which at best offer only an extremely restricted point of ingress to the emigrating cells, it was deemed advisable to employ elder-pith. The pith was cut into cubes of from 3 to 4 m. m., from which the outer and denser layer had been carefully removed. These cubes were first subjected to a temperature of 130° C. for about half an hour, then repeatedly boiled in a sterilized physiological salt solution, in which they were kept until required. It is important for the success of the experiment that all air be driven out of the pith cubes. In all experiments the piths were introduced into the peritoneal cavity. This offers many advantages, among which may be mentioned that the operation can be done without any bleeding whatsoever, so that the cubes are in no way defiled by blood, and that at the close of the experiment both the gross and microscopical changes can be more accurately observed in the peritoneum than in the subcutis. Ether was given for each operation. In no case was the animal at all inconvenienced by the foreign bodies in the abdominal cavity. All the rabbits kept perfectly well, and there never was the slightest trace of suppuration or infection or any abnormal reaction about the wound. In nearly all the experiments the piths remained in the abdomen for 24 hours, at the expiration of which the animal was killed by ether or piqûre, the abdomen reopened, and the cubes taken out and at once

dropped into the fixing solution. In each experiment from three to six cubes were employed, and these were fixed, some in saturated solution of bi-chloride of mercury, and others in Zenker's, the stronger Fleming, or Hermann's solution. From the fixative the cubes were taken through the regular routine, imbedded in paraffine, cut into thin sections, usually three to four mikromillimetres, and stained according to various methods: hæmatoxilin-cosin, methylene blue, both the ordinary alkaline solution as well as Unna's polychrome, Ehrlich's triacid, and others.

Before entering upon a short account of some of the experiments, it is necessary briefly to recall the several kinds of cells that will have to be considered. First the leukocytes. The rabbit [1] has polynuclear leukocytes which may be eosinophile or amphophile. These vary somewhat in their relative proportion under different conditions, but not sufficiently so as to require special attention in this investigation. The type of granulation has therefore not been considered, nor has it been deemed convenient to distinguish between polymorphonuclear and polynuclear cells. All these varieties will be discussed collectively as polynuclear leukocytes. In addition to the polynuclear cells there occur in the rabbit the large and small mononuclear leukocytes and the "Mastzellen." Besides these various forms of leukocytes there are cells which, as can confidently be stated, do not emigrate from the blood but from the tissues. Such are those large, more or less fusiform, sometimes more round cells, with a vesicular nucleus that does not stain as intensely in methylene blue as the nucleus of leukocytes, which contains one or two nucleoli and whose chromatin is arranged in a delicate network of filaments and granules. It is not necessary to give a detailed description of these cells and their behavior towards various stains. They have been most elaborately discussed by Ziegler,[2] Marchand, [3] and many others. These cells must be considered as originating from connective tissue and will be termed here simply tissue cells. There are other cells rounder and flatter, having a round or oval, rather large nucleus which contains no nucleolus and resembles in its behavior towards stains more the nucleus of the mononuclear leukocytes, though the arrangement of its chromatin is different. These cells resemble in many ways endothelial cells, though it is not possible to determine absolutely whether they are derivatives

[1] Hirschfeld: "Beitraege zur Vergleichenden Morphologie der Leukocyten." *Virchow's Archiv*, Bd. 149, 1897.

[2] "Experimentelle Untersuchungen ueber die Herkunft der Tuberkelelemente u. s. w." "Untersuchungen ueber die pathologische Bindegewebe- und Gefaessneubildung."

[3] "Untersuchungen ueber die Einheilung von Fremdkoerpern." *Ziegler's Beitraege*, Bd. ix., 1889.

of the endothelium. They will be called endothelioid cells. Lastly there are the plasma cells and all sorts of transition forms between the latter and mononuclear leukocytes. We will now give a brief epitome of some of the experiments.

Exp. 5. Large male rabbit ; four cubes of pith saturated with *acid* fluid introduced into the peritoneal cavity. The acid fluid was prepared by adding to sterilized distilled water sufficient of one-tenth normal sulphuric acid solution to just turn litmus paper. The whole was then again sterilized and the pith cubes saturated with the solution for several days. The cubes were placed in various parts of the peritoneum, the wound stitched up in two layers, the skin sutures covered with iodoform collodium. Operation entirely uneventful. Not a single drop of blood lost. After twenty-four hours the rabbit, which has been well and active, is killed with ether and abdomen reopened. Wound is found without reaction. All four cubes are found bunched together on the anterior surface of transverse colon, slightly adherent among themselves and to the peritoneum with a thin, fibrinous exudate. Small patches of very thin exudate on the peritoneum in the immediate vicinity of the cubes, as also a number of very small ecchymoses. The irritation is confined to the immediate vicinity of the cubes. All the remaining peritoneum entirely normal.

Microscopic Examination. The sections contain a great deal of fibrin, which is most abundant in the peripheral portion, but extends to the very centre of the cubes. All parts of the sections are filled with a multitude of polynuclear cells, many of which show unmistakable evidences of degeneration. Mononuclear cells are not numerous. Few tissue cells are found, these mostly in the peripheral portion and along the pith septa. Some have, however, penetrated far into the interior of the cubes. More numerous than tissue cells are the endothelioids. Plasma cells are also found, though not in great quantity. The entire picture is characterized by the great masses of polynuclear cells.

Exp. 6. Large female rabbit. Four cubes of elder-pith saturated with alkaline solution placed in different parts of peritoneal cavity. The alkaline solution was prepared by adding sufficient of one-tenth normal soda solution to sterilized distilled water to just turn litmus paper. Operation entirely uneventful. No bleeding. After twenty-four hours anima killed by piqûre and abdomen reopened. Piths not bunched together, but found in widely different places, and but very slightly adherent. Peritoneum everywhere entirely normal. No ecchymoses, no pseudo-membranes, no signs of irritation.

Microscopic Examination. A very considerable emigration to the very centre of the cubes. The overwhelming majority consists of polynuclear cells, but few of these show signs of degeneration. No mitoses. Moderate number of mononuclear cells both large and small. The mononuclears frequently lie together in groups. The tissue cells are very numerous. They are long and spindle-shaped, or more round or polymorphous, showing evident traces of amœboid changes of outline. Their protoplasm frequently contains vacuoles. They are found not only near the margin and along the pith septa, but also in the central portions of the sections. Endothelioid cells and plasma cells are also distinct, but not very numerous. Fibrin is present everywhere, but not in very great quantities.

Exp. 7. Male rabbit. Six cubes saturated with emulsion of staphylococcus pyogenes aureus in physiological saline (48-hours culture) placed in various parts of abdominal cavity. Operation uneventful. No blood. After twenty-four hours rabbit killed by piqûre and abdomen reopened. Three cubes are found bunched together on surface of transverse colon. They are imbedded in a firm white exudate, which offers some resistance to the removal of the cubes. After the piths have been taken out, the exudate remains firmly adherent to the gut with raised edges, reproducing mold-like the exact configuration of the cubes. The piths themselves are uniformly covered by the same firm white material. The other three piths are found single on the surface of different loops of intestine. All are

imbedded as described. In the immediate vicinity of the place of imbedding considerable hyperæmia and numerous small ecchymoses. Otherwise the peritoneum is entirely normal; no trace of fluid.

Microscopic Examination. A cursory glance shows nothing but great quantities of fibrin and enormous numbers of polynuclear cells. The sections to the very centre of the cubes are filled up principally with these. The majority of the polynuclears show distinct evidences of degeneration, thus presenting the ordinary microscopic picture of pus. They are in such abundance that it is difficult to make out anything else, even in very thin sections. Comparatively few mononuclears are found. There are some tissue cells, but they have apparrently not been able to penetrate very far into the interior of the cubes. They are found principally on the surface and the outermost layers, where they are gathered quite numerously. Plasma cells could not be made out.

Exp. 8. Large male rabbit. Six cubes saturated for several days in physiological saline (0.75), placed in various regions of abdomen. Operation uneventful. No bleeding. After 24 hours animal killed by piqûre. Four cubes found adherent together on a loop of ascending colon, imbedded in moderately firm, quite transparent, and colorless exudate. There is distinct hyperæmia, and irritation of the peritoneum in their immediate vicinity. One cube was found far down near descending colon, one other behind stomach. They were both well wrapped up in the same thin, glassy exudate, and adherent to peritoneum.

Microscopic Examination. Cell emigration not nearly as multitudinous as in previous experiment, though all the interspaces of the cubes to the very centre contain abundant cells. They are mainly polynuclear, only very few of which show signs of degeneration. Mononuclears are present, but not very abundant. No plasma cells can be made out. There is comparatively very little fibrin. Tissue cells are abundantly present, gathered mostly at the peripheric portions of the cubes and along the septa of the piths.

Exp. 9. Male rabbit. Four cubes saturated with a 48-hour bouillon culture of streptococcus pyogenes albus. Operation uneventful. No bleeding. After 24 hours animal, being perfectly well, killed by piqûre. Pith found in various places, principally in folds of colon, imbedded in deep masses of dense white exudate, similar to that produced by staphylococcus, only more bulky. Around the place of imbedding rather extensive hemorrhagic inflammations with ecchymoses and thin pseudomembranous exudate. All other parts of peritoneum normal. No fluid.

Microscopic Examination. Essentially the same as in preceding experiment.

Exp. 10. Male rabbit. Four cubes saturated with 2 % solution of sodium salicylate C. P. Operation as usual. After 24 hours animal killed by piqûre. One cube is found freely movable on surface of transverse colon. Three are wrapped up in large omentum. No visible signs of inflammation, apparently no exudate or any other reaction.

Microscopic Examination. Emigration moderate, but penetrating far into the interior of cubes. Overwhelming majority of cells polynuclear. Mostly well preserved. Only occasionally signs of degeneration. Large and small mononuclears occur. These are sometimes grouped together so that several successive microscopic fields would convey the impression that mononuclears were present in exceptionally large numbers. Then again wide areas would come into view that contain none. This same tendency of the mononuclears to travel in groups has been found in most of the experiments, and has been remarked by previous observers. Very few plasma cells are found. Tissue cells and endothelioids are quite numerous, not only along margin and septa, but also well in the interior of cubes. Comparatively little and thin fibrin. No mitoses.

Exp. 12. Male rabbit. Four cubes saturated with physiological saline (0.75), placed as usual in various parts of abdominal cavity. After four days animal, who has been entirely well and active, is killed by piqûre. On reopening abdomen, two piths are discovered slightly adherent to peritoneum of anterior wall of abdomen near the median line. Two imbedded in omentum behind stomach. No visible reaction of any kind anywhere.

Microscopic Examination. Very few leukocytes. In every field but a few, these mostly polynuclear with here and there a mononuclear usually of the lymphocyte type. The polynuclears show no signs of degeneration. The entire cube is taken up with tissue cells in active proliferation, and formation of granulation tissue. Numerous newly formed capillaries traverse the cube in all directions. It is beyond the scope of this paper to enter into a further description here. The picture is identical with those so exhaustively described by Ziegler, Marchand, Von Buengner,[1] and many others.

Exp. 13. Large male rabbit. Four cubes saturated with bouillon culture of streptococcus pyogenes albus placed within abdomen as usual. Rabbit, who has been lively and active and entirely normal, is killed after 48 hours by piqûre. Three cubes are found imbedded together on surface of colon in dense, hard, white mass of exudate. One cube imbedded in omentum is surrounded and rendered almost unrecognizable by a hard mass of coagulated blood, and white fibrinous exudate. No hemorrhages or other signs of irritation. Peritoneum otherwise entirely normal.

Microscopic Examination. Cubes are covered and filled with great quantities of fibrin, but the structure of the elder-pith is nevertheless discernible. Polynuclears crowd the field of vision in great quantity. They are mostly degenerated. Mononuclears, large and small, are very numerous. Here and there a sporadic plasma cell. Tissue cells are not numerous, and are found mostly at the margin and the outermost peripheral layers of the cubes. Endothelioids could not be made out with any certainty. No mitoses are seen.

Exp. 14. Male rabbit. Five cubes saturated with a 4 % formalin solution are introduced into the peritoneal cavity in the usual manner. Operation uneventful. After 24 hours animal, which has been entirely well and active, is killed by piqûre. Four cubes are found bunched together on surface of cœliac flexure of colon, imbedded in a soft, translucent, rather mucoid exudate. On the surface of colon and in immediate neighborhood of the cube numerous small ecchymoses. Peritoneum otherwise entirely normal. The fifth cube is found deep down among folds of small intestine, surrounded by similar exudate, but without ecchymoses.

The microscopic picture in this set is somewhat different from that found in the other experiments. The cubes are filled up in their entirety with cells, and there is comparatively little fibrin. The polynuclears are in the majority, showing but few traces of degeneration. The mononuclears, both large and small, with larger and smaller protoplasmatic bodies, are very numerous. Plasma cells are also quite frequent, and numerous transition stages between lymphocytes and plasma cells can be observed. Tissue cells and epithelioids are very abundant. Every field to the very centre of the cube contains these cells in large numbers. Mitoses are quite frequent. They are principally found in the polynuclears. There are besides occasionally "Mastzellen." Red blood corpuscles, which are sporadic in all the experiments, are here very frequently interspersed among the other cells in every field, even the most central.

Exp. 15. Male rabbit. Six cubes saturated with bichloride of mercury solution, (1 to 3000), placed in abdominal cavity as usual. Operation uneventful. After 24 hours rabbit, having been entirely well and active, is killed by piqûre. Piths found imbedded in hard, dense white exudate, much more bulky than in the experiments with staphylococcus and streptococcus. There are numerous ecchymoses in the vicinity. At the places where cubes are imbedded, loops of intestine are found matted to each other or to abdominal wall by the same white exudate. Rest of peritoneum is entirely normal.

Microscopic Examination. The cubes are completely filled with huge masses of cells and fibrin; the pith structure, barely visible, being covered over by the mass of cells and fibrin. The polynuclears are in overwhelming majority, showing all forms and kinds of degeneration, and presenting the picture of pure pus. Nevertheless an occasional mitosis can be distinctly recognized, though it remains doubtful whether it belongs to a polynuclear or a

[1] *Ziegler's Beitraege*, Bd. xix., 1896.

mononuclear cell. Large and small mononuclears are very numerous, but it takes some persistence to recognize them among the multitude of polynuclears. They frequently occur in groups, and thus seem more abundant than they really are. Plasma cells and their transition stages from lymphocytes not infrequent. Tissue cells and endothelioids present, but apparently in very limited numbers. It is possible that they may be covered over by leukocytes and their degenerative products, but the distinct impression is nevertheless conveyed that this type of cells is less numerous in this set than in the foregoing experiments. Altogether it is rather difficult to make out details amid the mass of polynuclears and their products of disintegration. Red blood corpuscles are plentiful everywhere.

A number of other substances have been tested in the same manner as the preceding, but inasmuch as they have led to essentially the same results, it is needless to detail them here.

Reviewing the foregoing experiments, it becomes evident that they tend entirely to corroborate the results of Borissow, insofar as no one of the substances tested has evinced any special affinity for any single type of leukocytes. In every instance the great majority of emigrating cells consisted of polynuclears. With perhaps one or two exceptions, pre-eminently the formalin set, the numbers of the polynuclear as compared with the mononuclear cells far exceeded their relative proportion in the normal circulating blood of the rabbit. This fact has already been noted by Borissow, and is again verified here. But while there is no evidence of any special affinity of any one substance for any particular form of leukocytes, the emigration of the latter as a body varies greatly in respect to quantity in the different experiments. While physiological saline and salicylate of soda caused but a very moderate emigration, while the alkaline fluid attracts less leukocytes than the acid, some, as staphylococcus and streptococcus, cause an enormous outpouring of leukocytes. In this connection it is interesting to note that of all the substances employed none, not even the bouillon cultures of the pyogenic bacteria, caused such excessive emigration and exudation as the ordinary 1 to 3000 solution of bichloride of mercury. The emigration and exudation caused by the bichloride can be designated both clinically and histologically as pure pus. But while the quantity of emigrating leukocytes varies according to the irritating properties of the substances employed, in no case has emigration been absent. A repellent action of any one of the substances as such, could not be established. Borissow found that physiological saline solution, for instance, has no attractive action on leukocytes. In our experiments with physiological saline we found very considerable leukocyte emigration. It has been asserted that emigration always, and under all conditions takes place into elder-pith, and that this is due to "tactile stimulation" of the leukocytes. It is difficult to conceive how tactile stimulation, if there is such a thing, can cause cells to

emigrate into the very centre of cubes of comparatively large size, such as have been employed in these experiments. I am rather inclined to side with Leber, who denies tactile irritation altogether, and attributes all emigration to chemical stimuli. Be that, however, as it may, it can safely be asserted that none of the substances here tested has shown evidence of any repellent action. Tissue cells of all varieties were found in every instance. Borissow has already noted the fact that these cells appear to be a regular component of pus in a rabbit. Von Buengner has stated that the tissue cells seem to follow more leisurely after the first voluminous onset of leukocytes has had time to subside. This is no doubt true, inasmuch as tissue cells were found in rather small numbers in those of our experiments with staphylococcus, streptococcus, and sublimate, in which the polynuclear emigration had attained large proportions. In those cases, too, the tissue cells have apparently not succeeded in penetrating very far into the interior of the cubes, but were chiefly collected around the margin and the peripheral layers of the pith. In other instances, however, where the leukocytes emigrated in no such rushing crowds, the tissue cells were distributed through the entire cube in fair proportion. This was especially the case in the formalin specimens. In no case, however, were they entirely absent. That their emigration was an active one and due to their own motion was apparent, not only because they were frequently found in the very centre of the cube, but also from the fact that their outline showed all stages and varieties of every possible amœboid change of form. The same remarks are true of the endothelioid cells. Karyokinetic figures were frequently found in both kinds of cells, not only on the outside of the cubes, but also in the interior. It is beyond the scope of the experiments briefly noted here to enter upon the numerous questions regarding the new formation of tissue, the phagocytic properties, and the various functions of the different types of cells. I cannot help remarking, however, that it was evident from these experiments, too, that the polynuclears take no part in the permanent new formation of tissue, but that this is the function of the tissue and endothelial cells. This could be particularly well studied in Experiment XII. In every case the polynuclears showed more or less signs of degeneration, the more so, the stronger the chemical irritant and the stormier their emigration. In Experiment XV., even after so short a period as 24 hours, the great majority of the polynuclears was entirely degenerated and to a considerable extent disintegrated. The polynuclears are transients, and this is so, though mitoses are not infrequently observed. As regards phagocytosis, it could be distinctly and frequently observed that the tissue and endothelioid

cells took up extraneous matter, such as broken-down leukocytes, red blood cells, cocci, etc. These experiments on the whole were not favorable to the study of plasma cells, inasmuch as these cells are known to develop more freely after the first 24 hours. Nevertheless at least a few could be made out, in most of the sections, and in several the transition stages between plasma cells and lymphocytes could be distinctly traced, thus confirming the recent statements of Marschalkó. It is outside of the limit of this paper to enter upon the subject, but it may be said, in passing, that there seems adequate basis in observed facts for the belief that plasma cells take part in the reparative process and in the formation of permanent tissue. If this is so, and their origin from lymphocytes must be accepted, as seems to be the case, then not all leukocyte emigration is merely transitory, and we must be prepared to regard a certain portion of permanent new-formed tissue as of possible hæmic origin. Lastly, it seems of interest to call attention to the peculiar behavior of the formalin as compared with sublimate. The latter causes an enormous reaction with a tremendous purulent exudate, while the former causes comparatively little irritation and the exudate is but slight. In the formalin sections the numerous tissue cells, lymphocytes, and plasma cells are particularly noteworthy. A further study of this substance, that has recently become so promising as an antiseptic and therapeutic agent, in its relation to cell emigration and tissue repair will undoubtedly prove of value.

SOME IMPORTANT ASPECTS CONNECTED WITH THE SCIENTIFIC FEEDING OF INFANTS.

By T. M. ROTCH, M.D.

MUCH credit belongs to those distinguished men whose continuous and earnest work has been so much quoted in the writings of Dr. A. Jacobi. Those of us, however, who have followed the contributions to the subject of infant feeding that have more or less permeated the literature of American medicine from the pen of Dr. Jacobi himself, must recognize the paramount influence which this distinguished author has maintained.

Three especial instances have always impressed the writer in connection with Dr. Jacobi's attitude toward this question. First, his singular willingness to fairly state and give credit to the opinions of others even when he differs from them. Second, his effort constantly employed to raise the question of infant feeding from the level of empiricism. Third, to bring the whole domain of infant life within the direct control of the physician himself.

I wish to draw especial attention to the last two phases of Dr. Jacobi's attitude toward this subject, as they appear to me to be of even greater importance than the opinion of any one man or any school of men.

Just as the other great branches of medicine and surgery have been withdrawn from the hands of charlatans and the laity, so should this branch of preventive medicine be placed on such a footing as to insure its scientific elucidation and that its administration be in the hands of those who best understand it.

In a paper of this limited scope it is impossible to discuss many of the points connected with this important subject. It will be impossible even to take notice of the various theoretical principles advocated by the leading pediatrists of the world on the question of the feeding of infants. For this reason, important as is the subject of direct and indirect maternal feeding it cannot be discussed here. I shall therefore limit this brief paper to a somewhat superficial discussion of certain

difficulties arising in and dangers to be avoided in prescribing and in modifying milk for the substitute feeding of infants.

Although in a certain number of instances it has been found that infants in the early months of life can digest and thrive on whole milk without any modification of the various elements of the milk, yet in so many cases this modification has been found to be necessary that it has assumed an important rôle in our practice.

In addition to the more common cases where some modification of cow's milk is required, there is a class, not infrequently met with, where even slight changes in the percentages of the elements of the milk and of the combination of these elements produce such marked effects as to impress upon us the importance of prescribing exactly.

Not only important, however, is the prescribing, but of still more importance is the assurance that the patient receives what is prescribed. It has happened that innumerable instances have come under my observation, where physicians have prescribed what was best, and yet where there was no doubt that a very different combination of percentages was actually given to the patient. In this way not only would unlooked-for results be obtained, but a very serious obstacle arise to our judging from experience whether the proper combination had been given for the especial case.

I have myself met with so many difficulties and dangers in prescribing a modification of milk, and have so frequently noticed how little these difficulties are appreciated, that it would seem wise and practical to bring some of the more important to the notice of those who are prescribing. Certainly if these difficulties and dangers are not recognized and avoided, the science of infant feeding cannot progress and must inevitably fall back to the domain of empiricism.

We must then acknowledge that the modification of milk for all ages, but especially in the early months of life, is extremely important, and that too much attention cannot be given to it by those whose practice makes it necessary for them to deal with the difficult problem of infant feeding. The modification of milk becomes an absolute necessity in a very large majority of cases, but this modification has many different phases and possibilities, and there is no doubt that improper and inexact modification may do untold harm. In quite a number of cases a somewhat macroscopic rather than a microscopic modification of the elements of the milk may be sufficient for the range of the individual digestion and for the nutrition of that especial case, but it has been my experience, in a very large number of infants whose vitality was low and whose malnutrition was excessive, to find that the lives of these infants could only be preserved by

gradual and minute changes in the percentages of the different elements of the milk and their combinations. We therefore cannot in a certain number of cases be too particular in assuring ourselves that we are using a milk modification which is as precise as our knowledge up to the present time has made it possible to be.

An improper modification of milk may be very unsafe for the patient, and in fact may lead to something worse than indigestion, namely infantile atrophy, rhachitis, or scorbutus. For instance, if we examine the food which was given in the earlier modifications of milk by simply diluting with water, we shall see why such dilutions failed to produce good results, simply because the nutritive quality of the milk was in large measure taken away from it by reducing the fat and sugar to such an extent that nutrition was impaired and rhachitis easily induced. After these simple dilutions with water came the many so-called "Cream Mixtures," which failed to produce the results expected from them, because the prescribers seldom knew what percentages or what combinations they were giving, having entirely overlooked the fact that there is no stable milk or cream, and that while they supposed they were using the same materials every day, they were in fact using many different materials which, combined in the same proportions every day, necessarily resulted in many different foods. For this reason, what might agree with the infant one day would disagree with it, unless by some peculiar good fortune, on many of the following days. My experience with these indefinite cream mixtures led me to investigate the cause of their failure, and forced upon me the conclusion that it was to a great degree the lack of determining for the special infant the percentages and combinations which that infant needed, and my conclusion was, that every infant must have its food adapted to its especial digestion and power of absorption, with resulting nutriment. This again led me to endeavor to perfect a means by which we could vary the percentages and combinations of the elements of milk to the greatest degree. Here I would state, as my experience may be of some possible value to other investigators on this subject, that in dealing with the proteids of cow's milk I have found that where the proteid digestion is weak, and in those cases where we ordinarily would have supposed that it would be well to predigest the proteids, I had greater success in adapting the proteids to the especial infant by starting with a very low percentage and gradually increasing the percentage up to a point which would be of the proper nutritive value to that especial digestion, than by attempting to predigest a high proteid.

As to the various artificially prepared foods which we unfortunately

still see in such abundance on the market, advertised in our best medical journals, and used as though they were the most inoffensive and useful means for relieving infantile gastro-enteric disturbance, rather than representing the opprobrium of intelligent and scientific physicians, there is nothing to say, except that they entirely fail to fulfil any of the practical and scientific factors of the feeding problem which they undertake to elucidate. To mention two instances out of the many which might be quoted of the total disagreement between the advertisement and the food as analyzed are the following:

A certain powder much advocated in certain quarters for predigesting the proteids of milk, and advertising that it supplies the deficient mineral matter when cow's milk is used in place of human milk, upon analysis is found to contain none of the mineral elements supposed to be lacking.

Another much-advertised cereal preparation, sold at a high price, shows by its analysis that it is nothing more than baked flour, the principal difference being in the cost per pound, as seen in the following table:

Advertised Cereal Preparation.	Wheat Flour (Average 25 Analyses).
Water	11.10	12.56
Ash	.38	.56
Albuminoids, including gluten	10.13	11.28
Fibre	.10	.27
Nitrogen, free extract	77.58	74.13
Fat	.82	1.20
Cost per pound	$1.00	$0.025–0.05

It can be truly said that these foods cannot be used rationally or safely since they so uniformly show a low percentage of fat and a bad combination of their other elements.

There is no question in my mind that, if milk is to be modified for the infant, it should be done under the physician's immediate supervision and exact prescription. Even when this is done, great and undoubted difficulties are met with. If we undertake to modify milk in the home of the patient, we may have numberless obstacles to overcome to obtain exact percentages and combinations, whether we use the ordinary commercial milk and cream, or the better cared-for milk and cream from special dairies, or milk and cream where the average percentages are known.

Let us consider some of the difficulties which occur in dealing with any method of modification. Supposing that the fat of such a milk is 5 %, and that in the mixture made from this material we make our calculations in such a way as to obtain 2 % of fat (in this case the milk is supposed to be from a Jersey cow), and the resulting proteids will

be 1.63. Again if we use the milk from a Holstein cow with milk containing only 3 % of fat, the resulting proteid will be 2.76, hence, unless an ascertained and accurately defined milk is used, the proteids may vary from 2.76 to 1.63, with the same prescribed fat of 2 %. These results are shown in the following table:

Milk Containing Fat.	Fat Prescribed.	Resulting Proteids.
5 %	2 %	1.63 %
3 %	2 %	2.76 %

As still further illustrating what different results regarding the percentage of the proteids may arise from using the various creams, I have had calculated and arranged the following table:

Cream to Work with.	Fat in Mixture.	Lowest Possible Proteids.
10 %	2 %	0.77
10 %	3 %	1.15
10 %	4 %	1.54
24 %	2 %	0.26
24 %	3 %	0.40
24 %	4 %	0.54
40 %	2 %	0.12
40 %	3 %	0.19
40 %	4 %	0.25

Here it is shown that unless the cream used is of known definite percentage, the mixture may show proteids varying from 0.12 to 1.50 as the lowest possible, even where the right quantity of cream is employed.

By studying these tables it will of course be seen that if a modification is indifferently done with reference to the quantity of the cream or milk employed, there will be serious differences in the percentages of all the constituents in the mixture, but if the same quantity of such a cream is employed without reference to its percentage of fat, there may be produced a radical difference in the food. It is therefore necessary not alone that the original formula shall affirm the exact cream to be used, but also that all changes be made by an experienced hand. Most persons, and among these qualified chemists, may easily forget the great difference in the results of a mixture, even when its ingredients do not differ very markedly. It is not strange, therefore, that the modifier should not know that the extra teaspoonful of cream or milk added to a formula mixture may change its whole character as to percentages, and of course as to its feeding value. Thus suppose that we have had made a mixture of twenty ounces with the percentages and changes as shown in the following table:

20-OUNCE MIXTURE.

(1) Fat 3 Sugar 6 Proteids 1
Adding a few tablespoonfuls of milk becomes
(2) Fat 3 Sugar 6 Proteids 2
Adding a little sugar becomes
(3) Fat 3 Sugar 12 Proteids 1

Here it is seen what changes a few tablespoonfuls of milk may produce when added to a 20-ounce mixture of a formula representing fat 3 %, sugar 6%, and proteids 1 %; namely, that the proteids have been raised from 1 to 2%. If we add to this same 20-ounce mixture a little of a certain sugar sold extensively as a fŏod, the sugar might at once be raised from 6 to 12 %. We also see how the addition of a small quantity of powder to a modified milk for the purpose of making it closely resemble mother's milk may raise the sugar so far above the percentage of any known breast milk. Also to substitute in modification a heavy cream for a light one, the difference not being discernible to the eye but only to the fat-tester, may change in a 20-ounce mixture a prescription for 4 % of fat to 10% of fat. For instance, if a formula is made up for the employment of 10% cream (top milk), and 20 ounces is prescribed for of the percentages of fat 4, sugar 7, proteids 1½, and in another day's mixture be put the same number of ounces of a similarly appearing cream but containing 24 % of fat, the change will be made from 4 % fat in the mixture to 10 % fat in the same mixture. In like manner when cane sugar is added, as is so often carelessly done to formula mixtures, the sugar may be doubled, and in some cases has been known to have been raised to even 30% in a home-made mixture.

In short, great accuracy is needed in employing the ingredients so as to produce the proper constituents, and we can, I think, formulate our ideas on this subject by saying that slight changes make great errors.

Although in a large proportion of cases the inaccuracy of milk modification results from the physician's lack of appreciation of exactly what material he is using, yet in many cases these inaccuracies are vastly increased by an insufficient oversight on his part when he orders the food to be mixed at home.

In quite a number of cases where the mixture is prepared by the mother she will do exactly as she is told, but as a rule the average mother is influenced by her maternal anxiety that her infant should have a plentiful and strong food; this means that she will add what seems to her a little more cream and milk than the prescribed proportions.

The evil results from the maternal breast-giving liberally whenever

the infant cries are well known, and this maternal solicitude operates disastrously when the infant is fed on prepared mixtures, for when it is probably additional water that is needed, it is additional milk or cream that is given. One of the many instances of this kind was where an anxious mother added a teaspoonful of cream to each feeding, thus changing the mixture from the 3% of fat intended to be given by the physician to about 6% of fat. The same argument applies to the careless use of measuring-instruments. The level teaspoonful or tablespoonful becomes the heaping one where the infant is concerned. A prominent physician in New York, who has had a wide experience with the modification of milk for infants, lately said: "I am afraid to let a mother know how weak a mixture I am using to save her baby's life, because I know she will overfeed it if she knows."

An insufficient knowledge of the materials used for modification arises first on account of the differences which are met with in the percentages of the materials used, and second on account of the unsafe character of the materials. These difficulties may be considered as one and treated in a general way as follows: The creams and milks of commerce are uncertain as to percentages and as to purity, but of these the "contract" milk is the most reliable and best cared for. The milk of most small dairies receives very poor care, and the diseases common to cows are concentrated in the small dairies supplied by a few cows. The contract milk, on the other hand, is a mixed milk, and the pathogenic organisms which may be present are attenuated by large dilutions. The single cow upon the private place, kept for the use of the family, is a great danger to home modification and also to the health of those who use her milk. It is curious to notice that nearly all the epidemics of contagious and infectious diseases that have been traced to milk have been so traced to small herds on small farms. It is self-evident, therefore, as there is such a great variety in the milks and creams which are obtained from various sources, that, if a modification is to be made with any approach to exactness in its percentages, the analysis of the especial milk and cream used each day is needed.

It is thus seen how important it is for the physician, if he decides to feed an infant on certain percentages, to avoid, by appreciating the difficulties which he must inevitably encounter, the danger of working with uncertain materials. He must appreciate that in making his calculations he can with certain percentages in the cream he is using only obtain a limited number of the percentages of the proteids, and that perhaps with the cream which he was using to obtain a certain percentage of proteids it would not be possible to obtain the decided change in the proteids which he might wish to give to the infant.

There is no doubt if these truths are kept in view that a formula can be used which is correct. However, we must remember that when we need to change the formula we must avail ourselves of a number of different creams, if the modifications are to be changed in cases where, as I have so often found, frequent and even great changes are needed.

I have spoken of the advantages of getting exact percentages; of equal importance is the use of safe milks and creams. A safe milk is not only free from germs of infectious diseases but also from a large number of bacteria of any kind, and from those flora of the bovine intestine which are so dangerous to infant life. This is a more serious matter than appears to the average thinker. While the milk laboratories may be entrusted to practically preserve the original purity of their milk supply during the process of its modification, yet it is especially in home modification where an originally clean milk may be rendered unsafe by careless work in the kitchen or in the nursery. Thus the handling of good milk may make it unsafe and bad.

In regard to the question of heating milk, sterilizing and pasteurizing milk in which certain pathogenic organisms have already developed their toxins does not destroy the chemical effect of these poisons. This is my chief protest against the use of unknown milks and creams, and the careless handling of good milk. Milk which has been full of bacteria and then has been sterilized may be dangerous because toxins of a poisonous nature may remain in it and act as poisons.

As to the use of bacteriologically unsafe milk for infant feeding, we should whenever possible know how good or how bad a milk we are obliged to use, if we expect to judge correctly of the success of our mixtures. We should endeavor to obtain a milk as free from bacteria as is possible in the especial case, and we can certainly be encouraged in our efforts in this direction by the recent reports which have been made on this subject.

Thus the Pepper Laboratory during the past year reports daily examinations from the Chestnut Hill Farm, in Philadelphia. The average for 334 days was 1530 bacteria per c. c., and this fell in the autumn and winter to 1150–1195 per c. c. When we take into consideration that 10,000 bacteria per c. c. is the maximum for good milk, it is clearly shown what an advance has been made in providing a pure milk supply.

A milk however is not only unsafe in a bacteriological sense; I have seen many instances where serious symptoms arose when a pure milk became dangerous from some unusual modification arising from a lack of knowledge of the modifying material used. In fact, unless we not

only know the percentage of fat in the cream which we use each day but also the possibilities as to obtaining with such a cream the other percentages such as that of the proteids, we are forced to prescribe more or less empirically. We also must run the risk as to whether we are dealing with an infant who may or may not be one especially susceptible to variations in the percentages and combinations of the elements of the milk in its food.

If the physician has decided that an infant is to be fed on some modification of milk, and believes that from time to time changes in the percentage of the mixture will be of value, he should recognize the dangers which may arise from an unsafe milk, and the difficulties of making correct calculations unless his materials are known each day.

Unless this is done, he will neither know, when the patient is doing well, what it is that is causing the improvement, nor, if doing badly, will he know the reason, and thus will not be able to intelligently make the proper changes.

ÜBER DAS VORKOMMEN VON DUCTUSGERÄUSCHEN BEI NEUGEBORNEN.

Von Professor Dr. THEODOR ESCHERICH, Graz.

DIE Anwesenheit eines lauten systolischen Herzgeräusches verbunden mit vergrösserter Herzdämpfung und ausgesprochener Cyanose bei einem Neugebornen dürfte wohl als genügend angesehen werden, um die klinische Diagnose eines congenitalen Herzfehlers zu sichern. Dass dies jedoch nicht unter allen Umständen zutreffend ist, zeigt der folgende, erst vor Kurzem von mir beobachtete Fall.

Schmelzer, Marie, wurde am 19. November etwas vor dem normalen Ende der Schwangerschaft in Gesichtslage geboren. Zwei Tage vor der Geburt war bei der Mutter auf der gynækologischen Klinik durch Herrn Prof. v. Rosthorn, dem ich auch diese Mittheilungen verdanke, eine Ovarialcyste durch Laparatomie entfernt worden. Der Wundverlauf war ein ganz normaler, jedoch wurden dabei ausgedehnte Unterbindungen von Gefässen vorgenommen. Die Geburt selbst verlief rasch, ohne Kunsthilfe. Das Kind war, obwohl keine erkennbare Ursache für Asphyxie vorlag, auffallend blau verfärbt, athmete oberflächlich und schrie mit schwacher Stimme. Es wurde deshalb schon eine Stunde nach der Geburt im Wagen in das Kinderspital überführt.

Die dort vorgenommene Untersuchung zeigte an dem ca. 2500 G. schweren, 45 Cm. langen, normal gebildeten Kinde die Zeichen der Frühreife. Die Athmung war sehr oberflächlich und frequent, das Kind lag ruhig ohne zu schreien mit mattem Ausdruck da. Sehr auffällig war die frequente, oberflächliche Respiration, die hochgradige Cyanose und blaurothe Verfärbung des Körpers, insbesondere des Gesichtes, sowie die Kühle der Extremitäten. Es war dies Veranlassung, sofort das Herz zu untersuchen. Es fand sich eine geringe Vergrösserung der Herzdämpfung, verbunden mit einem lauten systolischen Geräusch, das an der Herzbasis am deutlichsten, aber auch an anderen Stellen der Brustwand deutlich wahrnehmbar war. Der zweite Ton war rein, eine auffällige Accentuation desselben an der Pulmonalis nicht zu bemerken. Es wurde die Diagnose auf: congenitalen Herzfehler gestellt und das Kind sofort in die Couveuse gelegt. Im Laufe der nächsten Stunden entleerte es zweimal Harn, versuchte auch an der Amme zu trinken, jedoch nahm die Schwäche und Cyanose im Laufe des Nachmittags immer mehr zu, bis Nachts um 12 Uhr — elf Stunden nach der Aufnahme — der Exitus erfolgte.

Die Section ergab: Cyanotische Verfärbung der Hautdecken, Schädel mässig gross, Meningen sehr blutreich, von streifigen Blutungen durchsetzt, Hirnsubstanz weich und brüchig, Klein-Hirn pons und medulla auffallend blutreich. Thymus reicht bis zur Basis des Herzbeutels, nicht besonders vergrössert, dunkel gefärbt und von kleinen Blutpunkten durchsetzt, Anordnung der grossen Gefässe gehörig. Im fettarmen Herzbeutel ziemlich viel klare Flüssigkeit. Lungen zurückgesunken. Herz vergrössert. An der Vorderseite sind

beide Ventrikel, der rechte in der Ausdehnung von 4½, der linke von 2 Cm. vorliegend. In den Herzhöhlen dunkles, geronnenes Blut, die rechte Herzhöhle weit, ihre Wandung recht dick. Linkes Herz etwas kleiner, Klappen gehörig, Foranen ovale geschlossen, Ductus Botalli noch nicht geschlossen, verjüngt sich trichterförmig gegen die Einmündung in die Aorta, recht weit. Die Lungen sind zurückgesunken, nur in den hinteren Partien deutlich hepatisirt, blauroth verfärbt, glänzend, auf dem Querschnitte luftleer, sehr saftreich. Lagerung der Unterleibsorgane gehörig. Im Magen klarer, weisslicher Schleim, auf der Mucosa eine Anzahl runder, etwa linsengrosser oberflächlicher Erosionen mit dunklerem Grunde.

Dünndarm contrahirt, enthält dunkelgefärbten Inhalt; im Dickdarme meconium, Nabelgefässe gehörig.

Die Section hat somit im Wesentlichen eine ausgedehnte Pneumonie beider Unterlappen ergeben, die aller Wahrscheinlichkeit nach schon zur Zeit der Einleitung der Athmung bestand und mit der kurz vor der Geburt ausgeführten Operation in Zusammenhang zu bringen sein dürfte. Das Vorkommen solcher intra uterin erworbener Pneumonien wird von F. Weber bezeugt. Diese Affektion ist wohl auch die Ursache der hochgradigen Cyanose und des frühzeitigen Todes gewesen, der, wie die multiplen Blutungen erwiesen, unter Erscheinungen der Venostase und der Kohlensäure-Intoxication eintrat. Dagegen war das Herz, insbesondere die Klappen, vollkommen normal. Wie sollte man sich nunmehr das während des Lebens gehörte laute systolische Geräusch erklären? Die Annahme eines accidentellen Geräusches stösst gerade in diesem Lebensalter auf grosse Schwierigkeit. Ich möchte dem Satze Hochsinger's, der das Vorkommen desselben bis zum vierten Lebensjahre vollkommen leugnet, nicht unbedingt zustimmen. Kürzlich ist aus der Breslauer Klinik ein Fall mitgetheilt worden, in welchem der Sectionsbefund keine Erklärung für das im Leben gehörte Geräusch abgab. Auch glaube ich selbst über einen derartigen Fall zu verfügen. Allein hier fehlten alle jene Momente, welche zur Entstehung accidenteller Geräusche Veranlassung geben könnten. (Anaemie, Fieber, etc.) Auch der laute Charakter des Geräusches selbst widerspricht einer solchen Annahme. Die Localisation und das Intensivitäts-Maximum im zweiten Intercostalraum linkerseits sprach vielmehr dafür, dass dasselbe in den arteriellen Ostien oder im Ductus Botalli entstehe. In der That wurde der letztere bei der Section relativ weit und von der Pulmonalis gegen die Aorta zu verschmälert gefunden. Die kurz vor der Entbindung vorgenommene Auskultation der fötalen Herztöne hatte normale Verhältnisse ergeben.

Es spricht dies zusammen mit dem während des Lebens gehörten lauten schwirrenden Geräusch dafür, dass hier eine Persistenz des Ductus arteriosus Botalli und eine Durchströmung desselben von der Pulmonalis nach der Aorta vorhanden war.

Bei der Obliteration des Ductus wirken bekanntlich, wie durch die

Arbeiten von Kiwisch, Langer, Walkoffweber, Rokitansky, Schanz, festgestellt worden, eine ganze Reihe verschiedener Momente zusammen: die Ansaugung des Blutes, die Knickung und Zerrung des Ganges infolge der sich entfalteten Lunge, besondere Struckturverhältnisse der Wandung, Wucherung der Intima, Thrombose, etc. Wenn dadurch auch in der Regel der Verschluss derselben gleichzeitig mit dem Einsetzen der Athmung erfolgt, so ist doch nicht ausgeschlossen, dass auch im extra-uterinen Leben noch eine Zeit lang unter besonderen Druckverhältnissen ein Ueberströmen des Blutes aus der Pulmonalis in die Aorta stattfindet, da ja der Ductus regelmässig noch bis zur dritten Woche hin für eine Stecknadel durchgängig und erst am Ende des dritten Monats definitiv verschlossen gefunden wird. Es handelt sich hier um einen Rückfall in die zur Zeit des Fötallebens bestandene Blutcirculation, die von vielen Autoren als ein häufiges, ja physiologisches Vorkommniss angesehen wird. Von einem pathologischen Offenbleiben des Ductus spricht man nur dann, wenn dieser Zustand sich auch im späteren Leben dauernd erhält. In diesen Fällen, welche zur Gruppe der Entwicklungsfehler gerechnet werden, wird das Blut, wie Rokitansky gezeigt hat, von der Aorta nach der Pulmonalis getrieben und führt zur Entstehung eines lauten systolischen Geräusches, zur Hypertrophie und Dilatation des rechten Herzens und anderweitigen schweren Circulationsstörungen. Obzwar diese Vorkommnisse zu den seltenen Herzfehlern gehören (Rauchfuss), so besitzen sie trotzdem oder vielleicht gerade deshalb eine gute, durchgearbeitete Semiotik, um die sich Gerhardt, Schnitzler, Epstein, Hochsinger verdient gemacht haben.

Im Gegensatze hiezu haben die Fälle von einfacher Persistenz des Ductus Botalli von klinischer Seite noch sehr wenig Berücksichtigung gefunden. Es liegt dies daran, dass dieselben als physiologisches Vorkommniss aufgefasst wurden und dass man die Möglichkeit der Entstehung von Geräuschen nicht beachtet, ja vollständig in Abrede gestellt hat. Trotzdem scheint es mir nicht aussichtslos, der Diagnose dieses Zustandes etwas näher zu treten. Vielleicht, dass durch derartige Bemühungen das vage und unbefriedigende Krankheitsbild der angeborenen Lebensschwäche eine etwas schärfere Fassung erhält.

Wir dürfen erwarten, dass gerade die zu früh geborenen Kinder, bei denen die Aeste der Pulmonalarterien noch nicht ihre volle Ausbildung und Grösse erreicht haben, diesen Zustand am häufigsten und intensivsten aufweisen. Es ist möglich, dass die auffällige dauernde Cyanose, die oberflächliche zeitweise aussetzende Athmung, die Schlafsucht, welche diese Kinder zeigen, eben mit der ungenügenden Blutzufuhr zur Lunge infolge der erhaltenen Circulation durch den Ductus

Botalli in Zusammenhang ist. Jedoch ist in der Regel wenigstens bei der Auscultation kein Geräusch hörbar, weil einmal die durchtretende Blutmenge eine zu geringe Menge und Geschwindigkeit besitzt, und überhaupt beim jungen Kinde die physikalischen Bedingungen der Entstehung von Geräuschen wenig günstig sind.

Schnitzler, der auf das Vorkommen derselben geachtet, hat 100 Neugeborene darauf hin ohne positiven Erfolg untersucht, ebenso Duroriez. Auch sonst habe ich in der Litteratur keine Erwähnung gefunden. Nur Vierordt citirt in seinem neuesten Werke eine Angabe von F. B. Depaul (*Leçons des cliniques obstetricales*, Paris, 1872–1876), wonach das souffle fötal, das er bei 300 Untersuchten zweimal intergravididatem gehört hatte, auch noch extra uterin durch einige Tage zu hören war.

Unter diesen Umständen gewinnt unser Fall ein besonderes casuistisches Interesse, insoferne das durch die fötale Persistenz des Ductus hervorgerufene systoische Geräusch während des Lebens gehört, und der Nachweiss des offenen Ductus Botalli durch die Section erbracht worden ist. Leider ist die Untersuchung des Falles in vivo nicht so sorgfältig vorgenommen worden, wie es die Seltenheit des Vorkommens verdient hätte und ebenso ist die genaue Messung, sowie die Untersuchung der histologischen Beschaffenheit des Ductus unterblieben. Makroscopisch bot derselbe nur die gewöhnliche Länge, eine abnorme Weite und eine deutliche Verengerung gegen die Aortenmündung zu. Von sonstigen Veränderungen, welche mit dem Offenbleiben des Ductus Botalli in Zusammenhang gebracht werden, fand sich nur die Hepartisation der Lungen, die ja bei einem Neugeborenen an sich schon ein so seltenes Vorkommniss ist, dass sie von selbst sich zur Erklärung dieses Zustandes aufdrängt.

Die Frage, ob Hindernisse in der Respiration und im kleinen Kreislauf das Offenbleiben des Ductus begünstigen, ist vielfach erörtert worden. G. Weber war wohl der erste, welcher auf Grund klinischer Beobachtungen einen solchen Zusammenhang annahm. Ein Kind, das asphyktisch geboren und durch mehrere Wochen nach der Geburt wegen oberflächlicher Respiration mit kalten Begiessungen behandelt worden war, starb im Alter von $1\frac{1}{4}$ Jahren und zeigte einen weit offenen Ductus Botalli, nebst Hypertrophie beider Ventrikel. Bei einem zweiten, 3 Monate alten Kinde, das in den ersten Lebenstagen von einer Pleuropneumonie befallen war, wurde gleichfalls ein offener Ductus mit ausgesprochener Hypertrophie des rechten Herzens befunden. Virchow und Gerhardt schliessen sich dieser Anschauung an, die jedoch von Rokitansky in sehr entschiedener Weise bekämpft wurde. Der Widerspruch des Letzteren dürfte, wie Virchow bemerkt,

darauf zurückzuführen sein, dass Rokitansky nur die Präparate von älteren Personen mit pathologischer Persistenz des Ductus vor sich hatte. Ob man freilich mit Weber annehmen darf, dass diese letzteren aus den Fällen fötaler Persistenz sich heraus entwickeln, erscheint mir sehr fraglich. Jedenfalls sind die Momente welche zu einer vollständigen Behinderung der Obliteration und zum Einströmen des Aortenblutes in die Pulmonalis führen, ganz verschieden von denjenigen, welche hier in Frage stehen. Eine weitere Bedeutung des hier beschriebenen Falles scheint mir demnach darin zu liegen, dass er zu Gunsten jener Autoren spricht, welche geneigt sind, in der ausbleibenden Entfaltung der Lungen unter Drucksteigerung im kleinen Kreislauf, ein das Offenbleiben des Ductus begünstigendes Moment zu sehen. Da derartige Vorkommnisse, wie Atelektasen, Pneumonien, Fremdkörper in den Bronchien, etc., jedenfalls sehr viel öfter sich ereignen als Fälle von pathologischer Persistenz des Ductus im späteren Leben beobachtet werden, so ergiebt sich schon daraus, dass in den meisten derartigen Fällen mit dem Schwinden des Hindernisses in den Respirationsorganen auch der normale Blutkreislauf sich wieder herstellt und damit der Ductus, wenngleich verzögert, zur Obliteration und Involution kommt.

Eine willkommene Bestätigung dieser Anschauung bot mir die Beobachtung, die ich an einer wegen Debilitas vitae aufgenommenen Frühgeburt zu machen Gelegenheit hatte. Durch die Verbindung der Klinik mit der steierischen Landes-Findel Anstalt sind wir in der Lage, eine relativ grosse Zahl frühgeborener und lebensschwacher Kinder durch längere Zeit zu beobachten. Eine der auffälligsten Erscheinungen derselben ist die schon früher erwähnte oberflächliche, seichte Respiration, die oft so schwach ist, dass es besonderer Aufmerksamkeit bedarf, um sie zu zählen, oder überhaupt zu bemerken. Bei derartigen Kindern kann man eigenthümliche Anfälle von vollständigem Athemstillstand beobachten. Dieselben treten während oder nach dem Trinken, aber auch ohne jede äussere Veranlassung, anfangs seltener, später nur 1 bis 2mal des Tages, später aber mit zunehmender Schwäche des Kindes immer häufiger auf, bis schliesslich in einem solchen Anfalle der Tod des Kindes eintritt. Man erkennt dieselben par distance an der Zunahme der cyanotischen Färbung, die bei längerer Dauer der Respirationspausen die höchsten Grade erreichen kann. Das Gesicht und der ganze Körper werden schwarzblau, die Bulbi treten hervor, die Lippen schwellen an. Dabei werden die Extremitäten tonisch gebeugt oder führen mit angespannter Musculatur langsame, an Athetose erinnernde Bewegungen aus. Klonische Krämpfe habe ich dabei nie beobachtet. Bei längerer Dauer wird der anfangs beschleunigte

Herzschlag verlangsamt: es kommt zur Bildung der Asphyxia livida. Schliesslich wird das Kind blass, die Musculatur erschlafft, der Unterkiefer sinkt herab, das Kind macht den Eindruck als sei es bereits gestorben, weshalb auch diese Anfälle im Spitale den Namen der "Sterbanfälle" erhalten haben. Kommt nun spontan oder nach Application von Hautreizen, Einleitung der künstlichen Respiration, eventuell der Schultz'schen Schwingungen die Respiration wieder in Gang, so schwinden all diese Erscheinungen im Nu. Freilich dauerten bisweilen die Athempausen bis zu mehreren Minuten ohne dass die Herzaction sistirte. Es weist dies auf das geringe Sauerstoffbedürfniss der Kinder in der unmittelbar der Geburt folgenden Periode hin, während welcher sie, wie Schultze berichtet, noch 10 Minuten und länger nach Aussetzen der Nabelschnurpulsation zum Leben gebracht werden können. Es handelt sich hier meines Erachtens um ein auf nervöser Grundlage entstehendes, den Cheyne-Stokes'schen ähnliches Phänomen. Unter dem Einflusse der in Folge der oberflächlichen Respiration ungenügenden Sauerstoffzufuhr oder der langsam eintretenden Kohlensäure-Intoxication, wird das Athmungscentrum immer unempfindlicher, so dass die normale Reizschwelle nicht mehr genügt, die Inspiration auszulösen. Es kommt daher zu einer Athempause, welche entweder durch die künstlich eingeleitete Respiration oder durch den Erstickungsreiz der wachsenden Kohlensäure im Blute beendigt wird. Nach Einleitung der Athmung und Sauerstoffzufuhr zum Blute kehrt die normale Erregbarkeit für kürzere oder längere Zeit wieder. Ich habe diese, bei Frühgeborenen nicht seltenen Zufälle, in der deutschen Litteratur nur bei Epstein erwähnt gefunden, wohl aber ist eine von M. Henry mitgetheilte Beobachtung aus dem Pavillon des Debilles in Paris.

Ich habe schon erwähnt, dass es während dieser Anfälle in ähnlicher Weise wie bei der Asphyxia intra partum zur Verlangsamung der Herzschläge durch Vagusreizung kommen kann. In einem derartigen Falle, dessen Krankengeschichte und Sectionsbefund leider nicht mehr vorliegen, hatte ich Gelegenheit, jedesmal während des Athemstillstandes ein deutliches systolisches Geräusch in der Gegend der Pulmonalis zu hören. Dasselbe fehlte während der gewöhnlichen Respiration und im Beginne der Anfälle und war nur auf der Höhe der Asphyxie, alsdann aber constant durch eine Reihe von Herzschlägen, zu hören. Mit dem Eintritt der Athmung war es wieder vollständig verschwunden. Die Herzdämpfung war nicht wesentlich vergrössert, auffällige Cyanose nicht vorhanden. Ich glaube, dass es sich auch hier um ein Ductusgeräusch gehandelt hat. Die Athembewegungen haben bekanntlich einen mächtigen fördernden Einfluss auf die Circulation des Blutes in

der Lunge. Während des Athemstillstandes und in Folge desselben ist der Druck in der Pulmonalis erhöht. Dazu kommt vielleicht noch ein Absinken des arteriellen Druckes und diese beiden Momente zusammen bewirken diese Vermehrung der Druckdifferenz zwischen Pulmonalis und Aorta, so dass während dieser Zeit eine stärkere Durchströmung des Ductus Botalli stattfand und damit die Bedingung zur Geräuschbildung gegeben war. Mit Einleitung der Athmung kehren die Verhältnisse wieder zur Norm zurück. Diese Annahme scheint mir die plausibleste Erklärung für das intermittirende Auftreten dieses Geräusches. Uebrigens bedarf es auch hiebei noch der Annahme besonderer begünstigender Momente, da die Untersuchung anderer lebensschwachen Kinder während derartiger Anfälle zwar Asphyxie und Pulsverlangsamung, niemals aber ein derartiges Geräusch ergab.

Das Vorkommen dieser Ductusgeräusche hat nicht nur ein theoretisches, sondern auch ein praktisches Interesse. Der mit diesen Verhältnissen nicht vertraute Arzt kann ebenso, wie es uns mit dem zuerst beobachteten Falle ergieng, durch die Anwesenheit eines solchen Ductusgeräusches zur Annahme eines congenitalen Herzfehlers verleitet werden. Es wäre dies nicht nur ein diagnostischer Fehler, derselbe könnte auch zur Folge haben, dass der Arzt es unterlässt die unter Umständen indicierten und vielleicht lebensrettenden Methoden zur Anregung der Respiration energisch in Anwendung zu ziehen. Unter diesen scheinen mir die Schultze'schen Schwingungen am meisten empfehlenswerth. Nach Schanz kommt ihnen geradezu ein directer, den Verschluss des Ductus begünstigender Einfluss zu. Wir wenden dieselben auch bei älteren Kindern systematisch mehrmals des Tages an. Jedoch müssen sie bei frühgebornen, schwachen Kindern mit besonderer Vorsicht und ohne Gewaltanwendung ausgeführt werden. Es erscheint dies wirksamer und weniger grausam als der von Diemar (citiert bei Gerhardt) ertheilte Rath, blausüchtige mit offenem Ductus behaftete Säuglinge Tag und Nacht nicht zur Ruhe kommen zu lassen, sondern im Schreien zu erhalten.

IM TEXTE ERWÄHNTE ARBEITEN:

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ASPIRATION IN THE TREATMENT OF ACUTE TRAUMATIC EMPYEMA. WITH AN ILLUSTRATIVE CASE.

By JOSEPH D. BRYANT, M.D.

IN the brief considerations that are to follow we are disposed to speak of an isolated case falling under this topic, not only because of the medical interest it seems to possess, but chiefly because both the case and the means of cure were deeply interesting to the distinguished medical gentleman in whose garland of respect this contribution is but an unpretentious leaf. We are fully aware of the fact that an isolated example of an uncommon manner of practice is much more likely to be suggestive than convincing in its influence; if, however, it be but indicative of prospective utility, then indeed we are more than satisfied, and prompted to express the hope that it may be an earnest of greater outcome.

Inasmuch as the case in question is elucidative of the effect upon the thorax of blunt violence of a severe type, and also that the cases of this character are necessarily comparatively rare, a description of the accident along with its leading features will be given.

On July 26, 1899, a gentleman of about twenty-four years of age, and of good personal and family history, while sitting at the extreme and most exposed position on a trolley car going at a rapid rate, sustained a penetrating wound of the right side of the chest from the shaft of a heavy vehicle passing in the opposite direction. The end of the shaft entered the pleural cavity at a point just below the right nipple and corresponding to the 4th rib, passed backward between the 3d and 5th ribs to about their posterior third, and escaped at the right side, lacerating extensively the pectoral muscles.

The lateral portion of the 4th rib was broken into two fragments, each about four inches in length, the anterior fragment remaining attached to the 5th rib only by means of the intercostal tissues, while the posterior was connected to the same rib by a mere shred of these structures. The opening into the pleural cavity, therefore, was about eight inches in length and corresponded to the space between the 3d and 5th ribs, which were not injured. The integument covering the ribs was contused, especially in front, and torn for a distance of about four inches at the side. Hemorrhage had not been severe, and but slight shock followed the injury. Tympanitic percussion was manifest at each aspect of the right side of the thorax.

Under chloroform anæsthesia, and as soon after the accident as possible, the cutaneous wound was increased in size so as to permit a free inspection of the deeper portion of the injury. The anterior fragment of the rib was restored to its proper place and retained until union ensued by uniting together the borders of the torn tissues between it and the rib above with chromicized catgut. The posterior fragment was raised from within the pleural cavity, and, after severing its brief connection with the rib below, removed entirely. The muscular tissues at the side were greatly lacerated and the intermuscular planes filled with blood. The right lung, though markedly collapsed, was uninjured and projected partially from the opening during inspiration. Hæmoptysis was not present at this nor any subsequent time. The lower and posterior portions of the pleural cavity were filled with bloody fluid which freely escaped during deep inspirations. The fluid was poured from the cavity by turning the patient to the right side, and the space between the 3d and the 5th ribs was plugged firmly with iodoform gauze to permit of free cleansing of the soft parts without needless infection of the serous cavity.

Liberal incisions for drainage were then made, one into the axilla, the other at a point a few inches below, and the injured soft parts were freely laved with a solution of carbolic acid promptly followed by another, the hot saline solution. The soft parts were packed with iodoform gauze, the gauze between the ribs was removed, and the pleural cavity was freely flushed with hot saline solution. During the act of washing, the exposed surface of the lung was carefully observed, in order to detect appearances of injury, but none were found.

The inner and posterior boundaries of the cavity were exposed during expiration, and could be readily inspected. The convex surface of the diaphragm was explored with the finger for injury, and found to be intact. It is interesting in this connection to note that the anterior limit of the fracture corresponded to the attachment of the diaphragm at that situation to the fractured rib.

Four narrow strips of iodoform gauze were then introduced at intervals into the pleural cavity, the external ends hanging low without for drainage purposes. The iodoform gauze heretofore introduced to shield the soft parts during the washing of the pleural cavity was removed, rubber drainage tubes were carried through the openings, the entire inner surface of the wound was loosely packed with aseptic gauze, and the external wound and much of the cutaneous surfaces were covered loosely with a similar dressing, which was then held in place with adhesive plaster strips so applied as to impede as little as possible the respiratory movements of the opposite side of the chest.

It seems needless to add that the preparation of the field of operation and the entire subsequent technique were practised with aseptic care. It was hoped at the outset to prevent infection of the pleural cavity and thus obviate the occurrence of acute traumatic empyema and the subsequent happenings that its presence implies. But the nature of the injury, the character of the infecting agent and the fact that the clothing overlying the traumatism was torn, bloody, and had evidently been forced into the wound, forbade one the encouragement of a probable success. The wound was dressed for the first time on the third day and a specimen of the intrathoracic fluid was examined and pronounced sterile. On the fifth day, however, infective micro-organisms and pus corpuscles in the fluid were quite numerous. Suffice it to say that in spite of our best efforts free suppurative processes were soon fully established throughout the entire field of traumatism. The exposed surface of the lung was covered quite promptly with

a thick resisting layer of lymph which invested as well the parietal serosa. The purulent fluid was withdrawn from the chest by means of a syringe applied to the outer end of a rubber tube, the inner end of which was placed at the bottom of the suppurating cavity. Through the tube washing was done with boric acid solution after each withdrawal of purulent fluid. The wound was kept clean by a daily dressing of gauze. Because of the presence of the tube in the chest, it

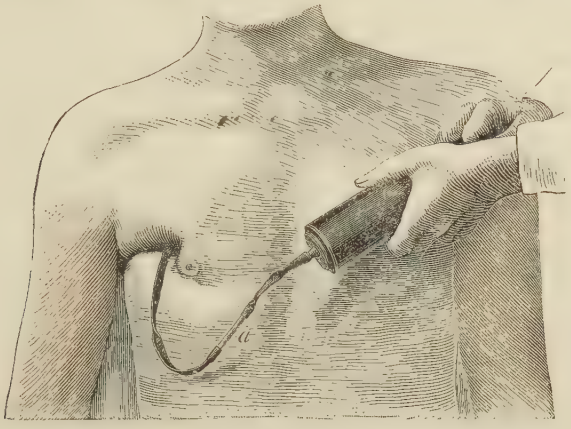

FIG. 1.

The Aspiration of the Cavity. *a*, the glass observation tube.

was easy to estimate, by measurement of the fluid introduced, the capacity of the morbid cavity. On August 3d it held sixteen ounces, about two ounces less than five days before. It was evident at this time that the expansion of the lung was lessening because of the restraining influence of the lymph covering its surface. Therefore it was determined to overcome this effect if practicable and at the same time to further facilitate the return of the lung to the normal capacity, by exhausting the air of the morbid cavity to a greater or less degree, thus lessening the atmospheric pressure from without. Accordingly various attempts were made to prevent the entrance of air at the sides of the tube following the pressure created by the withdrawal of air from the cavity. An aluminum shield, formed upon a mould of the wound and contiguous surface, and fitted with thumb-screws and other attachments, failed because of constant changes in contour of the chest wall incident to breathing, change of posture, etc., combined with the inflexible nature of the shield. Strips of adhesive plaster closely applied around the tube, and to the wound, reducing its size, were of fickle utility. Yet it can be properly said at this time that these failures were attended with commensurate good, for at first so trifling

a disturbance of air equilibrium as that produced by the removal of two drachms caused prompt and distressing cough, until uniform pressure was restored again by entrance of air. At a later period, complete exhaustion of air caused no responsive afflictions. The continuous and effective efforts of nature, combined with the abortive attempts of the writer, lessened the capacity seven ounces in twenty-four days, hence at the rate of two drachms and a third each day. Therefore on August 27th the cavity held nine ounces. The pulse,

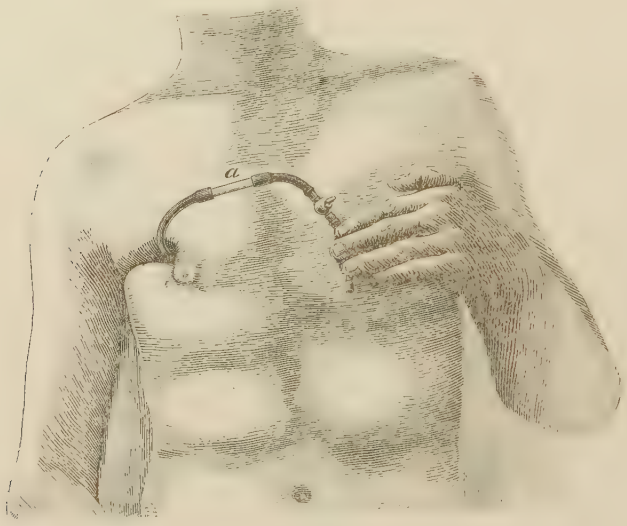

FIG. 2.

The Cavity Aspirated and Stop-cock closed to prevent admission of air.

temperature, and respiration differed but slightly from the average rate of the preceding four weeks, viz., 108, 101° F., and 25 respectively. At this date the withdrawal of air from the cavity with the syringe caused the soft parts to grasp the tube tightly and thus establish for the first time as complete and continuous a vacuum as was then advisable. (Fig. 1.) The attachment to the tube of a stop-cock made it possible to prevent the entrance of air after the syringe was removed. (Fig. 2.) Two days afterward a collapsed rubber bag was attached to the end of the tube for the purpose of maintaining a continuous vacuum when desirable. (Fig. 3.)

The curative action of this simple apparatus was easily obtained by exhausting the air from the cavity with a syringe, then closing the tube with the stop-cock before removal of the syringe, followed by the attachment to the end of the tube of a collapsed rubber bag to maintain the vacuum after reversal of the stop-cock. The space around

the tube where it entered the chest was closely packed with absorbent cotton saturated with carbolized oil to limit the entrance of air around the tube at that point. Several layers of aseptic gauze were placed over the field of injury and held in position by a body binder so fastened above as to prevent slipping down. The tube was held in place by safety-pins carried around it and fastened to the dressing. (Fig. 4.) Then the patient himself or the attendant had only to close the tube

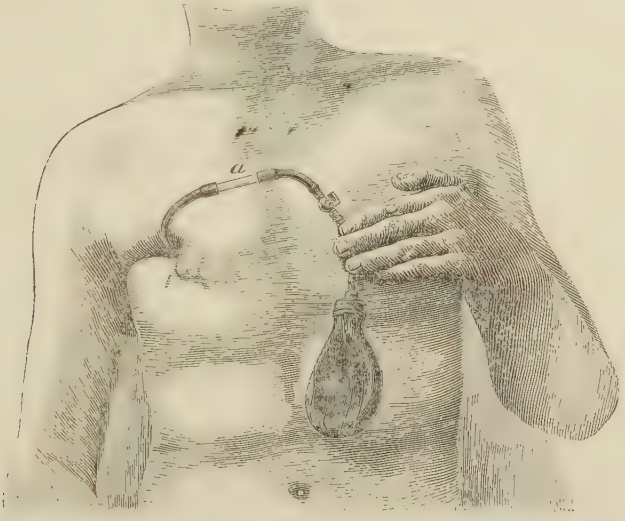

FIG. 3.

The collapsed rubber bag attached and Stop-cock opened ; the apparatus in action.

with the stop-cock before complete filling of the bag, expel the contents by squeezing, reapply it collapsed, reverse the stop-cock, thus reopening the tube, and again the apparatus resumed its action. Not only was air removed from the cavity by the suction but also a great proportion of the liquid inflammatory products as well. The introduction of a short glass tube (*a*) into a breach of the continuity of the rubber tube permitted observation, during the action of the bag, of the character of the fluid being withdrawn from the chest. As already stated, the cavity held on the 27th of August nine ounces; on August 31st it held six ounces; September 7th, two ounces; and on September 11th, when the patient was discharged, an ounce and a half, being a gain of seven ounces and a half in fifteen days, or an average of half an ounce a day, and therefore much in excess of the previous average.

The effect of the treatment on the pulse, temperature, respiration, and character of the fluid during the last two weeks of the treatment is noteworthy. The first week the pulse, temperature, and respiration

averaged 100, 101° F., and 22 respectively; during the last week, 85, 99° F., and 20 respectively. The character of the fluid during the first three or four days of this time changed but little, but during the last ten days altered gradually from a purulent to a serous nature. We hasten at this time to express the important fact that a too great exhaustion of the cavity of air during the earlier application of the plan caused an unpleasant sense of thoracic constriction and was followed promptly by sanguineous staining of the fluid withdrawn from

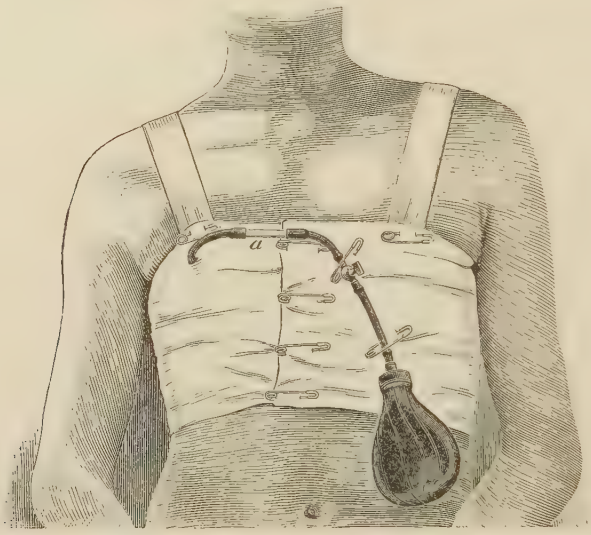

FIG. 4.

The dressings applied; apparatus held in place by safety-pins while in action.

the chest by the syringe. It was recognized that a too great degree of aspiration might not only cause undue determination of blood toward the cavity and objectionable bloody oozing into it, but also emphysema of the lung because of the general or localized excessive demand on the air cells arising from the consequent increased intravesicular pressure. Therefore aspiration forced to the extent of reddening the fluid was seldom practised, for it was regarded as entirely unnecessary and possibly unsafe. In fact the aspirating influence of the bag was kept at almost a minimum by close adjustment of the stop-cock regulating the calibre of the tube. After leaving our care on September 11th, the patient continued the use of the apparatus at varying intervals until the capacity was scarcely more than a drachm, and at the end of three months from the time of the injury the wound had substantially healed. A much earlier application of this principle of action could have been secured had it occurred to us to introduce into the opening when of much larger

size a tube suitably surrounded with an inflated rubber collar. (Fig. 5.) The width of the collar and the degree of the distension could have been easily arranged from time to time to conform with the requirements of the case. The one indicated in the illustration, although needlessly large and somewhat rude, answered the purpose well, clinging continuously and effectively for 24 hours to the thorax of a patient having a rigid walled cavity the sequel of an old thoracotomy. It seems entirely justifiable to assume that the obliteration of the cavity

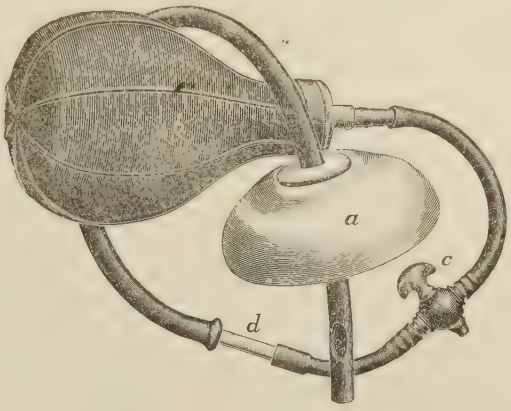

FIG. 5.

The Aspiration Apparatus. *a*, hollow rubber cushion; *b*, distended rubber bag; *c*, stop-cock; *d*, glass observation tube.

in the stated case was greatly facilitated by the preceding means, and it is also reasonable to suppose that prolonged suppuration, followed by much greater deformity than now exists, was prevented, and perhaps the radical measure of thoracotomy was forestalled.

As soon as this measure of treatment appeared to be entitled to seeming importance, we conferred with eminent practitioners of medicine regarding their opinion of its value, and inquired as to their knowledge of any previous utilization, for the purpose, of a similar means. From personal observation only, none were able to speak, but from belief based on recognized physical laws, and substantiated by this single instance, all were inclined to regard the method with rather more than favorable assent, in the treatment of recent acute cases. Realizing that a measure as simple as this could not possibly be regarded as entirely new, and expecting to learn from medical history of its conception and failure, or probably nothing at all because of its futility, we instituted a search of considerable extent of the literature of this topic. The results of the investigation disclosed, as one might

think, numerous instances of severe injury of the chest from penetrating violence of various kinds. Often indeed the extent and complications of the injuries were expressed in minute detail, to the exclusion of particular reference to the characteristics of the agent that caused them. We have been able to find but four cases of shaft injuries limited to the thorax, and two others of a similar nature. Doubtless many more such cases may be hidden in the literature, but they do not appear to have been collected by any writer, and in any event reports of this form of accident are for some reason comparatively rare. The chief object of search for that class of cases was to determine whether in any account the outcome differed practically from that in cases due to other causes. It was believed that a better standard of comparison with our own case could thus be established. Failing in this respect, the attention was then directed to the complications and sequels of chest injuries from penetrating violence, to ascertain regarding the employment in the treatment of acute traumatic empyema of any method akin to ours. In this connection we will venture to introduce the following.

Bouveret in his great work entitled *Traité de l'Empyème*, Paris, 1888, a work comprising nearly 900 pages, traces the history of the surgical treatment of this affection up to the date of the publication. It appears that the operation of thoracotomy goes back to the time of Hippocrates, but it was not until the XVIIth century that the trocar and cannula came into use. The operations of incision and puncture of the pleural sac, with injection of fluids, were the sole resources of the surgeon until 1859, when Chassagnac made us acquainted with surgical drainage.

About 1871 Potain introduced in the treatment the use of the siphon, which permitted the simultaneous evacuation and lavage of the pleural sac. At a little earlier period Dieulafoy, too, invented his aspirator.

In 1872 the Paris Academy of Medicine discussed the subject of empyema, and four methods of operative procedure were reviewed, viz.: thoracentesis, drainage, thoracotomy, and iodized injections. The operation of costal resection, originally suggested in 1869 by Simon, of Heidelberg, was performed for the first time by Estlander in 1877, although there appear to have been several claimants for priority, in particular Letievant, who claimed to have resected the ribs for empyema in 1875. If we may trust Bouveret, it thus appears that up to the time his work was issued (1888) no author had made any feature of seeking to radically cure empyema by persistently lowering the intrapleural pressure. Potain, who introduced the siphon,

may have expressed something of this idea, but his description, if ever published, is not accessible.

Bouveret appears to think that Estlander's operation was such a revolutionary advance in the direction of radically curing empyema that he recognizes no rival procedure.

Though Bulow was using siphon-drainage as far back as 1880, we have not succeeded in finding his original article; it is not mentioned in the catalogue of the Surgeon-General's library. His method appears to have been perfectly familiar to the members of the German Congress for Internal Medicine, held in 1890, for in the discussion on empyema at that time there are many allusions to his method of radically curing empyema by "permanent siphon-drainage employed for the purpose of producing negative pressure." Some of the members criticised the method adversely, which led Bulow to answer them in an article defending his procedure (*Zeitschr. f. klin. Med.*, Berlin, 1891, vol. xviii., p. 31). In this article he makes reference to the discussion on the treatment of empyema at the meeting of the Medical Congress at Vienna, 1891, in which sides were taken as to the relative advantages of siphon-drainage and resection of ribs. The majority adhered to the latter, and Bulow claims that this view is due to unfamiliarity with the method of siphon-drainage. The chief opponent of this plan appears to be Glaser, and Bulow's article is a refutation of the arguments of Glaser.

The latter states that the pretended advantage of siphon-drainage is that it produces an intrapleural vacuum during inspiratory efforts, thereby giving the lung an opportunity to expand. Bulow himself makes this claim, and has always reiterated that the chief advantage of this form of drainage is that it lowers the pressure within the pleural sac and thereby promotes the re-expansion of the partly compressed lung tissue. Glaser claims that the tight bandaging which is used after resection of the ribs conduces to the same end.

Bulow appears to believe that some confusion may have arisen because of the foolish claim of some advocate of siphon-drainage that a vacuum was produced thereby in the thorax rather than in the pleural cavity. He says that in an empyema which has been opened with trocar and cannula, the latter being left in situ, the intrathoracic pressure will be the same as the external atmospheric and intrapleural pressure! And the elastic fibres of the lung tending to cause the latter to retract, re-expansion is impossible. So long as the fistula is open, the pressure remains in equilibrium, subject only to slight variations incidental to inspiration and expiration. According to Bulow, this condition is distinctly unfavorable for recovery, and the rational indica-

tion is to produce negative pressure in the pleural sac in order to give the lung a chance to re-expand ; otherwise the patient is exposed to all the unpleasant sequels of empyema retraction of lung and chest, permanence of fistula, sepsis, amyloid disease, etc. To secure this "permanent aspiration" he employs the siphon-drainage, through which every inspiratory effort produces a sufficient amount of negative pressure to offset the elasticity of the lung. Wintrich found that 1/84 of the pressure of one atmosphere would offset this elasticity, while Donders makes the fraction 1/100. Bulow states that the pressure of a column of fluid 50-55 cm. high will fully offset this elasticity. If the pressure is more than this it causes intrapleural hemorrhage.

Bulow here introduces a case to illustrate the principle of his method. A chronic empyema (two years) was treated by incision and a cannula. Healing did not follow, and a freely discharging fistula resulted. The corresponding side of the thorax became much sunken and a uterine sound could be moved freely in the cavity in all directions. Bulow was about to send the patient to the surgical clinic for resection of the ribs, when the thought occurred to him that permanent aspiration of the pleural cavity might enable the lung to re-expand. A drain was introduced to the extent of 27 cm. and fitted to a siphon apparatus. After a space of three months the retraction of the lung had been considerably overcome, the cavity had become reduced to a mere fistulous tract, and the patient (who went about for a long time with the siphon bottle suspended from his chest) was finally cured by iodoform bougies introduced into the fistula.

This case shows plainly that lowering of the intrapleural pressure is a very practical method for securing the re-development of the lung ; and that after a period of inactivity of even 15 months a lung may regain its former usefulness.

Much of the objection to the plan of siphon-drainage appears to come from the belief that fibrin-coagula which often occur in empyema will clog the tube. It has been shown that this danger is largely imaginary and readily avoided by substituting a fresh catheter for the clogged one.

Perthes (*Verh. d. deutsch. Gesellschaft für Chirurgie*, 1898, p. 71) states that his method was made possible by the invention in 1869 of Bunsen's hydraulic pump which is in daily use in physiological and bacteriological laboratories. It produces a certain rarefaction of the air, and for use at the bedside a hydrant must be near at hand to supply the running water. The apparatus is ineffective unless the empyema-cavity is rendered air-tight—otherwise the external air will soon rush in and even-up the pressure. Perthes employed an inflated

cushion of soft rubber for this purpose, the drainage-tube passing through its centre. This cushion covered the thoracotomy incision, and was fastened in situ by a flannel roller. A vessel was employed in connection with the apparatus to collect and also measure the amount of discharge. A manometer applied to this collecting vessel indicated the exact amount of pressure within the pleural cavity. The pressure varies with inspiration and expiration, and as soon as the suction of the pump is applied the mercury rises in one limb of the manometer, so that when the desired degree of negative pressure is reached, the suction may be arrested.

Perthes has applied this apparatus in twelve cases of empyema. In the more recent cases auscultation and percussion showed that the lung had promptly re-expanded. In one case at least the ribs (6th and 7th) had been resected.

It was observed that after the aspiration a profuse serous discharge, often mixed with blood, preceded the appearance of pus in the reservoir. The author believes this fluid to have been forced out of the lymph spaces by the aspiration, and claims that it is salutary in that the poisonous products of the disease are thus flushed away. With regard to length of treatment, aspiration was kept up on an average for from seventeen to thirty days, while the entire treatment up to closure of the fistula ranged from forty-four to fifty days.

The degree of pressure which it is safe to employ is 10 cm. of mercury more or less. In a second case described at considerable length ribs had been resected by Trendelenberg. A third case is cited in which Bulow's siphon-drainage had failed and 5 cm. of the 7th rib were resected. In this case the aspiration was kept up forty-two days. In a fourth case 3 cm. of the 7th rib had been resected. According to this author the method is totally unlike siphon-drainage, because it is only a stage in the after-treatment of cases in which surgical interference has been employed. It is intended, however, to replace resection of the ribs, because it enables the lung to re-expand and adapt itself to the costal pleura, while in the operation of resection the costal pleura adapted itself to the collapsing lung.

Apropos of the reaction between positive and negative intrathoracic influences in the case of empyema the following is significant:

R. W. Parker in a discussion on empyema before the Royal Medical Society (*Lancet*, 1882, i., 689) quotes a plan advocated by Bouchut of forcibly overcoming the retraction of the lung by inflating the latter through a tube introduced directly into a bronchus. Parker regards this method as heroic, and proposed instead to displace the pleural fluid by introducing filtered and carbolized air into the sac, and

exhibited an apparatus devised for this purpose. He believed that after this procedure had been practised the air in the pleural sac ought to be less dense than the air within the lungs, and the intrapleural pressure less than the atmospheric pressure. This difference in pressure ought to be sufficient to enable the lung to re-expand; and would be maintained by the gradual absorption of the air within the pleural sac.

Other physicians present spoke favorably of the method. The theory appears to be as follows: the air introduced into the pleura tends to be absorbed somewhat rapidly. This absorption and rarefaction of air naturally lowers the intrapleural pressure and affords the retracted lung an opportunity to re-expand.

It appears from the foregoing that Potain in 1871, was the first to use the siphon-principle. Bulow seems to have been the first to develop the idea of permanent siphonage treatment, in the seventies.

Perthes, who employed the hydraulic air-pump, seems to have conceived the idea from Bulow's siphon-drainage method.

While it may not be to our credit, yet it is certainly true that the preceding method and the attending deductions are the products of our own conceptions. The comparative simplicity and security of the method commend it, but apparently it requires a more extended application to establish its practical utility. Neither time nor inclination prompts us to say more regarding it, as we prefer to leave its fate to the judgment of experience rather than to exploit its presumptive virtues even when sustained by immutable physical laws and by the practical demonstration which we have witnessed.

INFREQUENCE (ABSENCE?) OF ENDOCARDITIS AT NEW YORK FOUNDLING HOSPITAL.

By WILLIAM PERRY NORTHRUP, M.D.,

Professor of Pediatrics in the University and Bellevue Hospital Medical College; Attending Physician at New York Foundling, Presbyterian, and Willard Parker Hospitals; Consulting Physician to New York Infant Asylum; Member, Association of American Physicians.

IS endocarditis infrequent: is it absent?

Without encumbering a birthday celebration with too many figures, it will be our aim to give answers to these inquiries. The answers may add something to the accumulation of probabilities, which, after all, form the basis of the hourly decisions which constitute the responsibilities of the practising physician.

One who graduates from a general hospital is familiar to satiety with the lesions of nephritis, liver-cirrhosis, phthisis, and the like. If then such a graduate subsequently begins his pathological observations in an infants' hospital, he will turn as to a familiar face when he seeks to recognize in infants' lesions his old acquaintances of adult hospitals. With a mind full of such seeking after familiars, came to the work at the New York Foundling Hospital Dr. J. Lewis Smith, Dr. Jos. O'Dwyer, and the author of this paper. With such mental bias for examining the endocardium of infants coming to autopsy, it would be unlikely that any would escape routine examination. In fact, since 1873, and to my personal knowlege since 1882, the discussion has been ever new concerning the presence of endocarditis.

What has been the experience of heart murmurs? Have they been absent? In the experience of Drs. J. Lewis Smith, O'Dwyer, and myself have there been no physical signs which would call our attention to the lesion of heart valves? Yes, truly, and this has kept the discussion ever alive. There have been many cases in which the diagnosis has been made with some confidence. It has at times been accounted for by the presence of some developmental fault. But it is apart from any such accounting that the lesson here drawn is set forth. The

question has always stood thus before the trio: Has an undoubted case of endocarditis been diagnosticated in the Foundling Hospital in twenty-seven years? Has a case been discovered in the autopsy room in the same time?

Since we are avoiding figures, let us cite a frequent example of the search after this lesion. Dr. O'Dwyer being on duty, a note was sent to others, in some warmth of enthusiasm: "I believe I have a genuine case of endocarditis. It does not seem to me that a murmur so loud, so rough, so distinct, and so transmitted can be other than a sign of vegetations on valves. While under observation the sign has grown from nothing to a significant murmur, then louder, rougher, and more indicative."

To summarize the history of this illustrative case, I may add that the heart sounds were exceptionally pronounced, and I think for once the astute diagnostician, O'Dwyer, was led to commit himself to a diagnosis which he had eyed askance in that institution for twenty years. Autopsy followed, the child dying of a disease in no way connected with the heart or kidney; the *endocardium was normal*, the heart-valves were in no way roughened.

Since this must stand as an illustration, I may say that the examinations of all heart lesions have been by the methods in vogue at the time. Careful inspection may be claimed.

Here is our answer to the inquiry of frequency of endocarditis at the New York Foundling Asylum. I have told of the interest enlisted in the search, of the men of the time, Drs. Smith and O'Dwyer being two of them, and of the methods employed. Answer: In all the service of the New York Foundling Asylum of Drs. Smith, O'Dwyer, and myself, there never has been a single case of endocarditis recognized. This service embraces twenty-seven years of clinical observation, and between 2000 and 3000 autopsies.

I make haste to add that a well-known case of malignant ulcerative endocarditis has been reported in the New York Pathological Society, from the New York Foundling Hospital (Smith and Northrup). It is here referred to, but it is not what the reader will understand by simple endocarditis, a lesion compatible with continued life. It is rather a septicæmia. It is the only case of endocardial lesion recorded, and serves rather to accentuate the rarity of endocarditis. It is scarcely the form of this disease one expects to find when searching for endocarditis, from previous experience in general hospitals for adults.

It may be parenthetically added that rheumatism is as rare as endocarditis at the hospital, though the records have not been studied with care as to this point. Chorea is rare.

Summary: Endocarditis at the New York Foundling Hospital is infrequent or absent.

Clinically: Heart-murmurs have been noted many times. These have been transitory, anæmic, ultimately recovering, or been unexplained by autopsy findings.

At Autopsy, or Clinically: There has not been a case of undoubted endocarditis at the Foundling Hospital in the combined experience of Drs. J. Lewis Smith, Jos. O'Dwyer, and the writer, from 1873 to 1900.

Exception noted in a single case of malignant ulcerative endocarditis, reported in New York Pathological Society Proceedings. [See Reports of N. Y. Pathological Society, 1884.]

Rheumatism is rare.

Chorea is rare.

Ages of cases under observation, four years and under.

From October 1, 1873, to January 1, 1900, 27,278 infants have been admitted to the care of the institution. Autopsies to the number of 2000 to 3000 have been recorded.

IODIDE OF IRON IN THE TREATMENT OF CERTAIN FORMS OF INFECTIVE ARTHRITIS.

By J. C. WILSON, M.D., Philadelphia.

THE treatment of certain forms of infective arthritis, especially gonorrheal arthritis, is notoriously unsatisfactory. The salicylates are useless either to relieve the pain or to bring the process to an end. Iodide of potassium is little better. Rest, measures to improve the nutrition, and local treatment including fixation of the joints and, later, massage and Swedish movements are rational procedures which do not, however, prevent disability and suffering, often protracted for months. Quinine, iron, and arsenic are prescribed on general principles. The treatment by dry air at high temperatures is occasionally but by no means always followed by good results. It is furthermore not available in all cases. Surgical measures, such as incision and drainage, are also sometimes useful.

For several years I have treated these cases with large doses of the syrup of the iodide of iron with satisfactory results. Recently several gentlemen associated with me in clinical work have adopted this plan and bear testimony to its usefulness. A considerable number of successful cases could be collected from my hospital service and private practice in support of the plan. The clinical phenomena are, however, so variable that the mere marshalling of statistics may be misleading.

The following cases recently observed are fair examples:

Case 1. Susie S., eighteen years of age, German, unmarried, a waitress; admitted to the German Hospital, September 25, 1899. No member of her family has suffered from rheumatism. Excepting the diseases of childhood she has always had good health. Menstruation regular and normal; leucorrhœa at times; no other pelvic symptoms. Denies exposure to danger of venereal infection. Pain in right shoulder and ankles, which are also slightly swollen and tender upon pressure. Moderate elevation of temperature; no sweating. Was treated with salicylates and alkalies. Discharged October 21st, at her own request, improved. A diagnosis of acute rheumatism was made.

The patient was re-admitted to the hospital on the 25th of October, four days later, suffering from intense pain in the right shoulder and right knee. Moderate swelling; no redness; exquisite tenderness. Signs of considerable intra-articular effusion in the knee. There was an elevation of temperature—102° to 104° F.—of sub-continuous type. The

patient's general condition was wretched. Every movement was attended with intense exacerbation of pain in the affected joints. Appetite poor. Bowels constipated. Sleep prevented by pain except after the use of hypodermic injections of morphia. The cardiac impulse feeble; not visible, but palpable in the fifth intercostal space just within the mid-clavicular line. A soft systolic murmur at the base. Marked venous hum on the right side.

Marked Leucocytosis, Gradually Decreasing.

Nov. 8,	hæmoglobin	65 %,	erythrocytes	5,150,000,	leucocytes	31,200
" 18	"	49 %	"	4,070,000	"	23,200
" 23	"	61 %	"	4,780,000	"	9,200
" 29	"	61 %	"	5,580,000	"	7,200

On the 8th of November the knee was aspirated and a clear straw-colored fluid obtained which yielded upon culture a growth of staphylococci but no gonococci. On the same date an examination of the pudenda revealed an inflammatory condition of the mucous membrane with great redness and tenderness and a copious whitish discharge. The cover slip preparations from this discharge failed to show the presence of gonococci. The hymen was absent.

During the course of the case, namely, on November 4th, a splotchy erythematous rash appeared, first upon the face and neck, then upon the arms and hands, later upon the body and lower extremities. This eruption was not attended by pain or itching. On the third day after its appearance some of the spots became petechial and two days later many petechiæ showed themselves. This rash gradually faded out and had disappeared by November 19th. It was attributed to the sodium salicylate.

The urine was markedly albuminous and contained epithelial, hyaline, and granular casts and an excess of indican. At the time of the patient's discharge the urine contained a trace of albumin but no casts.

The treatment in this case from the time of admission until November 9th, a period of sixteen days, consisted in the free administration for a time of sodium salicylate in combination with sodium bicarbonate; later of sodium salicylate alone, 6 grammes, afterwards increased to 9 grammes, in twenty-four hours, in connection with phenacetin. Later oil of wintergreen was given in full doses. The local symptoms were unaffected by this treatment and the general condition became steadily worse until November 9th, from which time all other medication was stopped and the patient was given syrup of the iodide of iron in increasing doses. The dose was at first 2. t. d. In the course of a day or two the dose was increased to 3. t. d. and again shortly to 4. t. d., and finally to 4. q. die; and with this dosage, practically a teaspoonful four times a day, the medicine was continued until the patient's discharge from the hospital.

Within a few days from the beginning of this treatment there was notable improvement not only in the general condition but in the arthritis. The temperature gradually fell and on November 21st reached the normal, from which it did not again rise. By the 19th the eruption had wholly disappeared. Upon the 20th it was noted that the peri-articular effusion is no longer present. Prior to this, pain and tenderness had ceased and in the course of a few days the function of the affected joints was restored and the patient was able to be out of bed. On the 20th a 10 per cent. ichthyol ointment was applied to the knee joint and continued for some days.

Case 2. John H., twenty-two years of age, a chairmaker by occupation, was admitted to my ward in the Pennsylvania Hospital September 24, 1899. The patient stated that he had suffered from inflammation of the stomach when a child, but had otherwise had good health. Had never had rheumatism. Had used alcohol at times to excess. Had gonorrhœa about a year ago, the discharge lasting about two months. Since that time the patient had not been in very good health. About two weeks prior to admission began to feel tired and suffered from headache; had some diarrhœa and was unable to work. Presently there developed

stiffness of the joints with great pain, which prevented him from sleeping. The first joint implicated was the left knee; later the shoulders, the other knee, and the joints of the fingers. Upon admission the patient had fever, restlessness, and delirium. Tongue dry and brown, with sordes; moderate cardiac hypertrophy; faint systolic murmur in mitral area and at base, not transmitted; some tympany without abdominal tenderness. Knee joints much swollen, tender, and painful, without redness. The other affected joints somewhat better than at first. Urine acid, 1018; contained pus and large numbers of red blood corpuscles; albumin, probably due to the blood.

The treatment consisted at first of salophen .6 q. q. h. together with sodium bromide 1.3, the local application of soap liniment and oil of gaultheria to the joints. A few days later this was exchanged for an alkaline treatment, without advantage. On the 2d of October he was given salophen again in larger doses, and later potassium iodide. His condition up to this had remained about the same. On the 4th of October it was noted that his mental condition had improved, but on the 6th his condition was again worse, with delirium and much complaint of pain in the knees and through the head and neck. The former treatment was stopped and patient was given syrup of the iodide of iron m. xxx, q. die, to be increased m. i. each day. The dosage was progressively increased until the 5th of November, when the patient took somewhat more than a drachm of the syrup of the iodide of iron four times daily without repugnance or the slightest derangement of the digestion. Meanwhile his condition had steadily improved. By the end of October the temperature had fallen to normal and the patient was about the ward on crutches. He was discharged entirely recovered on the 17th of November.

Case 3. David D., twenty-one years old, a clerk, was admitted to my service in the Pennsylvania Hospital October 19, 1899. Had gonorrhœa three years ago, during the course of which there was pain in the left shoulder and in both feet. These pains continued eight months and have occasionally recurred. Two weeks ago a second attack of gonorrhœa occurred, which continues. Was admitted to the hospital on account of severe pain in both feet, with great tenderness and swelling and inability to walk. A purulent urethral discharge with blood. Was treated with strontium salicylate .6 sext. die, and salol. In the course of a few days a slight improvement resulted. On October 26th it was noted that the pain in the feet was very severe and that the urethral discharge had ceased. He was given syrup of the iodide of iron in doses m. x, t. d. Two days later no improvement. The syrup of the iodide of iron was increased to m. xx, t. d. At this time there were pain, tenderness, and swelling in the right knee. November 5th, given the syrup of the iodide of iron m. xxx, t. d., the dose to be increased by the addition of m. i daily. On the 8th, condition generally improving. On the 15th, improvement in the general condition and in the joints; able to walk about the ward. On the 28th was discharged cured. In this case the constitutional disturbances were slight and the temperature ranged between 97° and 100° F. during his sojourn in the hospital.

These cases not only illustrate the curative influences of the syrup of the iodide of iron, but they also place the results of treatment by this medicament in striking contrast with the failure of ordinary antirheumatic remedies such as the alkalies, the salicylates, salophen, and potassium iodide.

In Cases 1 and 2 the treatment was conducted in my absence for a considerable period of time by a medical friend temporarily in charge of the wards. It was not until my return to the German Hospital early in November that Case 1 was treated with the iodide of iron and the same change was made in Case 2 upon my taking the service in the Pennsylvania Hospital about the first of October. In Case 3 other

measures of treatment were at first designedly employed in order to place the iodide of iron treatment in contrast with them.

Gonococci were not present in the vaginal discharge in Case 1, and doubt as to the essential nature of the infection naturally arises. There is no question, however, as to the character of the infection in Cases 2 and 3.

It would be idle to speculate as to the process by which the therapeutic effect is produced. The subject belongs to the wide realm of empirical therapeutics. Favorable results have frequently been obtained in the convalescent cases by the substitution of gelatine-coated pills of the iodide of iron for the syrup, but in the acute cases the pills are of inferior value.

A NEW METHOD OF DETECTING AND DETERMINING GLUCOSE IN URINE.

By Dr. J. RUDISCH,

Attending Physician Mount Sinai Hospital, New York.

PROFESSOR HUPPERT, in his classical work[1], in discussing the method of detecting glucose in the urine, comes to the conclusion that small quantities of glucose can not be detected with absolute certainty by any single chemical test or combination of such tests. In such cases, he recommends making use of the polariscope, provided the instrument is sufficiently sensitive. If the urine turns the plane of polarization to the left (several minutes in a decimeter tube), then dextrose is not present; if not, the result is doubtful. In case of dextro-rotation, the urine very probably contains sugar. In order to be perfectly sure, the urine is fermented for half a day, at a temperature of 34° C. in a water bath, is decolorized by means of acetate of lead, and once more examined. The disappearance of dextro-rotation, or appearance of lævo-rotation, in a urine previously optically inactive, can only be attributed to the presence of sugar. A clearly evident excess in the amount of gas produced by urine in contact with yeast, compared with the gas produced by yeast in contact with water alone, indicates the presence of sugar. Just as in the case of polarization, Huppert recommends fermentation as a control for the other reaction. If the reducing substance disappears, then that substance is sugar.

In my opinion, the following weighty objections can be raised to Huppert's conclusions. First, for very small amounts of sugar, an excellent instrument and a great deal of practice are the first requisites; and even then the presence of lævo-rotating substances in urine may vitiate the results considerably. Second, the reducing substances in urine cannot be entirely eliminated by fermentation. The following table shows that filtration through animal charcoal and subsequent fermentation are not efficient for this purpose.

[1] *Analyse des Harns*, 1898.

TABLE 1.

Before Fermentation.	After Fermentation.	Before Fermentation.	After Fermentation.
0.17%	0.05%	0.10%	0.06%
0.10%	0.04%	0.09%	0.08%
0.09%	0.07%	0.03%	0.02%
0.11%	0.05%	1.40%	0.08%
0.12%	0.06%	2.40%	0.07%
0.12%	0.06%		

From one of Huppert's conclusions — drawn from a thorough study of the subject and confirmed by all investigators — it follows that there is but one reaction characteristic of glucose—that is, its conversion into carbonic acid and alcohol by the biological process of fermentation in contact with yeast; and since this conversion is but partial, and varies with concentration and temperature, it can indicate only approximately the amount of sugar present.

An ideal way of determining the presence and quantity of sugar in a given specimen of urine would be a method of extracting it, so that it could be identified physically, and weighed and measured as such. This line of research has been followed by many investigators, but the practical results have been unsatisfactory, as it requires a great deal of chemical knowledge and apparatus, experimental ability, and the expenditure of no little time. After a thorough trial of all the proposed methods, by means of which I rarely succeeded in obtaining more than from 0.5 to 0.75% of the sugar actually present, this method was abandoned.

As the physician is often confronted with the question of the presence and amount of sugar in urine, a rapid and exact method is essential. From the preceding introductory remarks, it is evident that the utilization of the property of fermentation possessed by sugar gives the most hope of a certain answer to the question of its presence in urine.

As has been already stated, in the process of fermentation principally carbonic acid and alcohol are produced, together with a number of other substances having no distinctive reactions, which vary in quantity with the conditions under which fermentation takes place, so that they are useless as a test of the presence of sugar. The two main products have, however, undoubted characteristic reactions enabling them to be detected when only small quantities are present.

The detection of alcohol seemed to me at first to be the easier, and to this end the following procedure was adopted: The urine was acidified with sulphuric acid, and boiled briskly for ten minutes, so as to drive off any traces of acetone, aldehyde, and diacetic acid which might

be present, and which, answering to the same tests as alcohol, would interfere with its detection. The urine was then neutralized, fermented with yeast, and distilled. If now potassium hydroxide and Lugol's solution were added, iodoform was produced if alcohol was present, and could be easily recognized by its characteristic smell and yellow crystals. In the distillation of urine, however, compounds are formed, partly due to the fatty acids of urine and partly to the volatile substances formed by fermentation, which are capable of masking the smell of iodoform. It was therefore concluded, that the presence or absence of alcohol could not be positively ascertained, except after redistillation and considerable further trouble. In my hands, after taking all precautions, sugar in urine containing less than 0.25 % could not be detected with certainty, and with benzoyl chloride the results were even less satisfactory. For quantitative purposes, this method was manifestly unsuitable.

Of the many methods proposed for the detection and estimation of sugar, directly or indirectly, depending on the carbonic acid generated, I shall mention but two, because after careful consideration they appear to me to be the best. These are: Einhorn's fermentation test, and Roberts's differential-density method. Both are empirical, and of the two, Einhorn's is better adapted for the detection and estimation of small quantities, while Roberts's, unsuitable for this purpose, is especially applicable to urines containing more than 1 % of sugar. Einhorn's belief, that 0.1 % is easily detected by the saccharometer, and even 0.05 % is not beyond the range of possibility, under favorable conditions, is an opinion not shared by many. In my own use of this method, the limit was reached at 0.15 %, and even then the results were not always absolutely convincing. In the employment of Roberts's method, in order to reach an exact determination, careful consideration of temperature and very accurate instruments are required; then, too, it is scarcely applicable to those cases where the need of a positive answer is pressing.

I then tried to determine the carbonic acid in fermented urine, by removing the gas by means of an air pump. My modus operandi was as follows: An ordinary stout flask was partially filled with urine, the air in the flask was then exhausted, and a stream of air deprived of carbonic acid was then passed through, and this process continued until I was unable to detect free carbonic acid in the urine, by passing the air exhausted from it through a solution of caustic baryta. Next a small quantity of yeast[1] was added to the urine, and, the flask being

[1] Fleischmann's yeast was always used; a cake weighed about twelve grammes.

tightly closed, fermentation was allowed to go on for several hours at a temperature between 30° and 35° C. Then the flask containing the urine was connected with an absorption bottle containing barium hydroxide, and the air exhausted through it. The presence of carbonic acid was shown by the turbidity of the baryta, and the amount subsequently estimated by a standard acid.

By means of this method, it was demonstrated that yeast in a watery solution fermented at a pressure of from two to three inches of mercury, half a cake giving as much carbonic acid as would correspond to fifteen milligrammes of sugar. It was also found that the amount of carbonic acid did not always correspond to the mass of yeast used.

To prevent autophagy of the yeast cells, *i. e.*, self-fermentation, which, as was shown by Jodlbauer and others, always takes place when there is a great amount of yeast present as compared with the sugar, I added only one-half as much yeast by weight as there was sugar, to the various urines and watery solutions. Under these conditions, fermentation proceeded very slowly, so that — to give an example — only one-half the sugar of a 0.1 % solution was used up in eighteen hours, a result very unsatisfactory when the question of time was so important.

From the fact that urine contains a small quantity of unfermentable substances, which reduce copper sulphate in alkaline solutions, it will be evident that the only reliable method of detecting glucose is a titration before and after fermentation. I shall give here a description of the principle upon which my method is based.

If, for example, 10 c. c. of urine reduce 10 c. c. of Fehling's solution, and, after fermentation, the same amount of urine reduces but 5 c. c. of the solution, it is evident that in the unfermented urine there was sugar enough to reduce 5 c. c. of Fehling's solution, and we can say that $\frac{1}{4}$ % of sugar was originally present, as 1 c. c. of Fehling's solution reduces 5 milligrammes of sugar. Fehling's solution is, however, not easily applicable to the detection of small quantities of sugar in the hands of the inexperienced, as the end reaction is uncertain, requires a practised eye, and repeated titrations are necessary to be sure of the result. To obviate these very weighty objections, I had recourse first to Pavy's modification of Fehling's solution, but abandoned it for the following reasons: First, the titration must be performed rapidly, or the ammonia is given off, and the cuprous oxide is precipitated. Second, the presence of tartrates is objectionable, as the solution is liable to deteriorate on standing. Third, the strong yellow color, due to the action of the caustic alkali present, on the sugar, interferes materially with the end reaction.

After investigating a great many modifications of the above solutions,

the following was found to be exceedingly satisfactory. The solution consisted of:[1]

Copper sulphate (crystallized),	4.78	gms.
Sodium sulphite,	50.	"
Sodium carbonate (crystallized),	80.	"

Dissolve in 10% ammonia water and make up to 500 c. c. One c. c. of this solution is decolorized by 1 milligramme of sugar. The solution was standardized against pure glucose crystallized from alcohol, and the double salt of glucose and sodium chloride.

The glucose reduces the blue solution of cupric oxide to a colorless solution of cuprous oxide. The latter has a great affinity for oxygen, and if it is in free communication with the outside air, is easily oxidized to cupric oxide. This, if not prevented, will vitiate the results of titration. Another point to be considered is that, on boiling, ammonia is given off, which makes it unpleasant for the operator. To remedy these deficiencies, the following apparatus was devised:

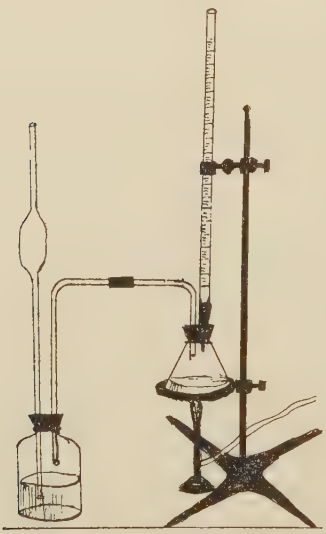

FIG. A.

The Erlenmeyer flask containing the standard solution is fitted with a double-bored cork. Through one hole a tube leads to the burette, through the other to the absorption apparatus containing a sufficient amount of 10% sulphuric acid to cover the exit tube, so that it is sealed from the outside air.

My modus operandi is as follows: 10 or 20 c. c. of my standard solution are put in the Erlenmeyer flask, diluted with water to 50 c. c., and heated. When boiling briskly, the urine, decolorized by animal charcoal (*vide infra*), is allowed to drop from the burette until the blue color disappears. When a large amount of sugar is present, the titration is better performed with diluted urine. The following results were obtained by

[1] The amount of sodium sulphite, sodium carbonate, and ammonia may be varied greatly. A solution that gives identical results with the one here described, and which is more easily extemporized by the druggist, is the following:

Fehling's solution of copper sulphate, 69 c. c.
Saturated solution of sulphite of soda, 200 c. c.
Carbonate of soda, 80 grs., dissolved in 200 c. c., 10% ammonia.
Make up to 500 with ammonia water (10%).

analyzing liquids containing known amounts of sugar. A glance at the tables will show the accuracy of the results.

In Table 1, known amounts of sugar were added to water; in Table 2, known amounts of sugar were added to fermented and decolorized urine:

TABLE 2.

Added Per cent.	Found Per cent.
0.05	0.05
0.10	0.10
0.25	0.25
0.50	0.53
1.00	1.01
2.00	2.04.

TABLE 3.

Added Per cent.	Found Per cent.
0.15	0.17
0.30	0.28
0.50	0.53
1.00	1.05
2.00	2.07

FIG. B.

For practical purposes, the following method will be found convenient, satisfactory and rapid: from 1 to 10 c.c. of urine filtered through charcoal if necessary, are put into an Erlenmeyer flask, diluted to about 100 c.c. with water, boiled and titrated with the above solution from a Binks's burette (of which a cut is given) until the blue color remains after two minutes' boiling. On account of the unavoidable oxidation of the cuprous oxide by the oxygen of the air, a little more copper is required for the reaction, the difference being about 10%. Knowing this, and adding 10% to the result found, an accurate result will be obtained. In other words, by this method 1 c.c. equals 1.1 milligrammes sugar. The following table will show the accuracy of the method: known amounts of sugar were added to decolorized and fermented urine.

TABLE 4.

Added Per cent.	Found Per cent.
0.25	0.24
0.17	0.17
0.07	0.05
0.08	0.06

The advantages of my modification of Fehling's solution over others proposed are:

First: It is more stable than Pavy's solution, as the titre was unchanged after four weeks.

Second: Sodium carbonate decomposes sugar much less rapidly than sodium hydroxide, and the resulting color is not such a deep yellow.

Third: The sodium sulphite shortens the titration process considerably.

Fourth: One can titrate with this solution directly into the diluted urine, as above described. Fehling's and Pavy's solutions cannot be used in this fashion, as they destroy sugar so quickly as to lead to an absolutely false result and show less sugar than there really is present.

In titrating urine, the end reaction is often obscured by its yellow color, so that it is difficult to be sure of the titre within two or three cubic centimetres. Lead acetate could not be used for decolorizing the urine, on account of the presence of sodium carbonate in my solution, as the resulting precipitate of lead carbonate would obscure the reaction. I was at first deterred from using the most efficient way of decolorizing urine — by means of animal charcoal — by the fact that so many authorities had found sugar retained by it. An article of Roberts's[1] encouraged me to go over this field again. The results were most gratifying, as in no case was an appreciable amount of sugar absorbed by the charcoal. The different results obtained by other experimenters and by myself may be due to our different methods of using the charcoal. While they placed the charcoal on a filter paper in a funnel, and filtered the urine through it, I shook four volumes of the urine with about one volume of charcoal, let it stand for half an hour, and filtered. The solution was then generally colorless, but in a few cases the addition of more charcoal and longer standing were necessary. Another advantage of the filtration was the removal of reducing substances, such as uric acid and urates. The results obtained in urine and water solutions are given in the following tables.

Table 5 shows the result of titration of solutions of pure glucose in water before and after filtration through charcoal; the figures in Table 6 were obtained in the following manner: The urine was fermented for twenty-four hours so as to deprive it of glucose, different quantities of glucose were added to different samples, and the titre determined before and after filtering through animal charcoal, as above described.

TABLE 5.

Before Fil.	After Fil.
Per cent.	Per cent.
0.05	0.05
0.10	0.10
0.25	0.24
0.53	0.52
1.00	1.01
2.04	2.06

TABLE 6.

Before Fil.	After Fil.
Per cent.	Per cent.
0.17	0.15
0.28	0.30
0.53	0.54
1.05	0.99
2.07	2.03
0.26	0.27
0.50	0.51
1.10	1.04
1.50	1.56
2.10	2.05

[1] *Practitioner*, 56.

In order to shorten the time of fermentation, large quantities of yeast (one-quarter to one-half a cake) were added to from 30 to 50 c. c. of the urine, and the bottles kept in a bath at a temperature of from 30° to 40° C. It was thus found that a large quantity of sugar was destroyed in one-half an hour, and in a few hours but little or no sugar remained. The results given in Table 7 were obtained in the following manner: The urine was fermented for twenty-four hours, and filtered through charcoal, glucose was added, and it was then allowed to ferment with yeast. The tables give the amounts of sugar left after various periods of time, at a temperature of 30° C.

TABLE 7.

Time (in hours).	Sugar Present.	Time (in hours).	Sugar Present.
0	0.35	1½	0.15
½	0.25	24	0.06
1½	0.14		
2½	0.12	0	2.10
3½	0.08	½	1.13
4½	0.06	1	0.66
24	0.05	2	0.32
		3	0.20
0	0.25	24	0.13
½	0.17		
1½	0.13	0	3.60
24	0.05	½	1.50
		1	0.22
0	0.30	2	0.12
¾	0.18	24	0.10[1]

From the above tables, we see that for the detection of sugar one-half hour fermentation is sufficient. For quantitative purposes, four hours will be sufficient to give a satisfactory result, and for an exact determination eighteen hours are necessary.

From my experiments, I have come to the following conclusions:

1. No single chemical reaction nor combination of them can be relied upon for the detection of small quantities of sugar in urine.

2. Cases may arise where even the presence of a moderate amount of sugar may be masked by other substances, so that the detection of it by chemical tests alone is difficult, and sometimes impossible.

3. The only characteristic reaction of dextrose is the property of fermentation in contact with yeast.

4. The detection of sugar in urine by testing the alcohol produced

[1] As to the figures for "sugar present" after twenty-four hours, in the tables, these do not represent sugar residues, as the urines possessed the same amount of reducing power after being fermented for another twenty-four hours. They represent the amount of reducing substances, other than sugar, present in the urines, even after decolorization with charcoal.

by fermentation, either with benzoyl chloride or by formation of iodoform, is complicated and uncertain when small quantities are concerned.

5. The detection of the presence of glucose by the carbonic acid produced by fermentation, either by Einhorn's or by my (vacuum) method, is neither a quick nor a certain way for small quantities, and Einhorn's, of these two methods, is the easier and better.

6. Titration in an apparatus with exclusion of air is as accurate and can be done as quickly as with Pavy's solution, and my solution has the advantage of not changing its strength after standing for eight weeks.

7. The determination of the amount of sugar by titrating with my solution into the diluted urine with a Binks's burette is accurate enough for all purposes required by a physician, and can be done more quickly than by any other method.

8. By my method of double titration, it can be determined that sugar is present in from one-half to one hour, even if only 0.1 % is present; within three hours a sufficiently accurate determination of the whole quantity can be made, and an absolutely correct one at the end of twenty-four hours.

9. My solution can be used by the practitioner for the ordinary determination of sugar, as Fehling's has been (without the fermentation), and possesses the advantages over the latter that I have mentioned in the body of the article.

My thanks are due to Dr. J. Waddell, Mr. D. McIntosh, and Dr. A. G. Foord, for their help in carrying on these investigations.

I. TOXIC PEMPHIGUS OF A NEW-BORN CHILD.
II. GENERAL INFANTILE ATROPHY AND THE INJECTIONS OF SERUM.
III. GANGRENA PULMONAR DIFUSA LATENTE.

By Dr. A. MARTINEZ VARGAS,

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I.

TOXIC PEMPHIGUS OF A NEW-BORN CHILD.

PEMPHIGUS of recently born children gives rise to many problems, which have not as yet been solved in a clear and definite manner.

This disease is usually considered to be of microbic origin on account of the germs which have been discovered in the liquid contents of the bullæ. In perfect accordance with this theory and from our study of the numerous cases observed in asylums, lying-in hospitals, and private practice, pemphigus is considered to be contagious, infectious, at times epidemic, and even of malignant severity. In fact, Glatz has claimed that the name pemphigus should not be applied to pemphigoid eruptions of a mild character, which are easily cured. On the other hand, cases of this disease are described, the dermic elements and bullæ of which rest upon a but slightly inflamed base or even upon healthy skin and yet are preceded and accompanied by malaise, fever, disorders of the digestive organs, and other general symptoms comprising pemphigoid fever. A syphilitic variety of pemphigus is recognized, the localization of which on the soles of the feet and palms of the hands determines the dermatological variety and the predisposing conditions. There is also a cachectic variety, and others following scarlet fever, measles, lichen, and prurigo, or at least concurrent with these diseases. In short, as a more rational and efficient treatment of the process, we prescribe that which has for its basis the reconstructents — especially arsenic.

The case of pemphigus which I shall describe in the following

remarks can not be included in any of the groups of which I have just given an outline, because it was not of microbic origin nor was it caused by contagion or infection. It was mild, since it was not followed by fever or general indisposition and was cured in a few days by very simple local treatment. In my opinion this was a case of toxic pemphigus produced by ptomaine poisoning of the mother during the later days of pregnancy.

On account of its mildness, the simplicity of its symptomatic manifestations, and the efficiency of local treatment, this case of pemphigus resembles the variety described by A. Jacobi on pages 5 and 6 of his very important work, *Therapeutics of Infancy and Childhood*,[1] rather than the description given at the beginning of this paper, which is the one found in most of the text-books.

The clinical record is as follows: In the Obstetrical Clinic of this University there was born, after normal accouchement, on the 27th of April, 1899, a female child with every appearance of perfect development in respect to size, weight, and freedom from all deformity. Thirty-six hours after birth, or during the afternoon of the second day, without having given sufficient evidence of indisposition to attract the attention of the mother, the child's skin began to be covered with round stains of a reddish color which, a few hours later, were converted into vesicles or blebs, so that on the third day after birth the child presented the clinical picture of pemphigus. Two days after, *i. e.*, on the fifth day, we took charge of this little patient, who then presented the appearance shown in the adjoined photograph. The eruption was then confluent on the cheeks and chin. On the neck, arms, forearms, backs of the hands, and the right pectoral region we discovered very numerous vesicles and bullæ, while the conjunctivæ, the buccal mucous membrane, the palms of the hands, and the soles of the feet were wholly free, showing not the slightest trace of the eruption.

In the midst of a mass of vesicles were several bulky detached blebs $\frac{3}{8}$ of an inch (1 cm.) in diameter, the largest being in the left superciliary region, on the shoulders and around the elbow. The blebs, as well as the vesicles, were circular or oval in shape, having a thin semi-transparent covering formed by the horny layer of the epidermis. The contents was at the onset clear and watery, but after the lapse of a few hours it became cloudy and grew opalescent with a slightly blood-stained hue. The tension of the liquid was intense, and on making a few slight prickings with a needle there came from the vesicle several small drops. A few of the bullæ appeared shrivelled,

[1] Philadelphia, 1898.

A Case of Toxic Pemphigus.

others had already dried up, either on account of spontaneous rupture or having been broken by contact with the linen, so that the blebs were torn open and presented a flat surface with a border or rim of epidermis. The cuticle in these areas was pearl-colored and doubled upon itself in folds, resembling the vesicles produced by cantharides or burns of the second degree. In places the dermis was denuded and red, yielding a small quantity of serum. The skin upon which these vesicles appeared was reddened and erythematous over a small area, but wherever there was an interval free from eruption the skin was absolutely normal. The liquid gave an alkaline reaction, and the bacteriological examination was entirely negative, no germs of any kind being found. A few inoculations were made into the healthy skin with a sterilized lancet without causing infection.

On the whole the state of the child was satisfactory; the temperature did not exceed 37.3° C., the digestive apparatus was in a healthy condition, and the child took the breast freely and slept soundly.

On account of the mildness of the case I prescribed a purely local treatment, merely dusting with bismuth subnitrate and protecting some regions with cotton. Under this treatment the bullæ and vesicles soon dried up and the ulcers healed without scar, not even an incrustation or other blemish remaining, and on the 6th of May both mother and child left the Clinic in good health. No other case of pemphigus appeared in the Infirmary in spite of the fact that there were several other new-born children in the ward with but one nurse to bathe and dress them all.

On investigating the causes which might have given rise to this case, contagion was excluded, for there was no other case either in the lying-in ward or those adjacent to it. In fact it is many years since a case of pemphigus has been seen in this ward. There is no reason for attributing it to syphilis, for in addition to the absence of its characteristic occurrence on the palms of the hands and the soles of the feet, there were no other evidences of this disease upon the child, nor was there any taint in either parent. The cachectic variety and those of measles, scarlet fever, lichen, and prurigo are, of course, excluded. Having eliminated these causes, I questioned the mother in regard to her diet, when she replied that, in consequence of poverty, she had frequently been without food; that during the last three months of her pregnancy there had been many days when she had been unable to procure nourishment, and that during this period her diet had been limited to sprats, herrings, and more or less black pudding. To this wretched food it was very seldom that she was able to add even a dish of beans. Almost invariably her meals consisted of two or three salted

sprats, sometimes eating six or eight in the course of the day. Occasionally she suffered from diarrhœa, headache, and some eruption on her arms, which was accompanied by itching.

From the investigations of Gautier and Etard, Bochlisch, Arnstamoff, Brieger, Anrep, and Ehremberg, we know that in fish in a state of putrefaction and in salted herrings various toxic bases are to be found: hydrocolidine, escombrine, parvoline, neuridine, etilenediamine, muscarine, gardinine, methylgardinine, trietilamine, putrescine, dimethylamine, ptomatropine, and ptomatomuscarine, and that, although the symptoms produced by these toxic substances are not absolutely characteristic, it is known that hydrocolidine causes shivering, tetanic convulsions, and death, the heart stopping in diastole. The best known and most toxic of these substances are the ptomatomuscarine of Brieger and the ptomatropine of Anrep; the latter in doses of two milligrams produces in the rabbit midriasis, convulsions, and death by cardiac paralysis. Later Griffiths discovered sardinine, $C_{11}H_{11}NO_2$, in sprats in a state of putrefaction, a substance capable of causing death with vomiting and diarrhœa. Likewise Ehremberg found in dried sausages the same ptomaine of substances in a state of putrefaction, and Nauwerk found a bacillus which possesses the property of decomposing albumin.

From these facts it is quite logical to judge that during the period of three months, while the mother was nourished in a manner so extremely wretched and opposed to systemic requirements, she must have absorbed some of these more or less toxic bases into her system. This conclusion is justified by the frequent attacks of diarrhœa and the occasional erythema and itching of the arm from which she suffered. It was, in fact, a kind of toxic dermatitis, admitted by the majority of authorities at the present time to be a well defined variety of this affection. On the other hand, we know, from demonstrations of an experimental nature, that toxic substances contained in the blood of the mother pass to that of the fœtus in the same way as microorganisms do, as proven clinically and experimentally. We also have proof that the converse is true—that is to say, that a poison injected into the blood of the fœtus is capable of intoxicating the mother, provided the circulation through the umbilical cord is not interrupted. Therefore the same substance which produced the eruption in the mother reached the blood of the fœtus, remained in its blood, and at the moment of birth elimination of this poison occurred through the vascular skin, which cutaneous congestion is present in every new-born child, and the eruption was the same as had already occurred on the skin of the mother.

Another physiological fact must be borne in mind—the delicacy of

the skin in all new-born children, shown by the desquamation which always takes place. This must have had such an influence upon the elimination of the toxic product that, instead of causing an erythema or other simple form of dermatitis, the irritation was so severe as to cause pemphigus, a more complex variety, since it is accompanied by a great transudation of serum.

In order that my interpretation may be justified, the case under consideration must be regarded as one of toxic pemphigus of a new-born child, caused by poisoning of the mother through her food, and therefore we should agree upon a new variety of this affection, which we might classify in the following manner:

- Acute pemphigus of the new-born child..
  - Idiopathic...
    - Toxic.... — The liquid of the bullæ is sterile. The disease is produced by intoxication of the mother by improper food or bacteria. It is not contagious or epidemic.
    - Microbic.. — The liquid of the bullæ contains one or more varieties of microbes. The disease is due to contagion and is contagious in turn.
  - Deuteropathic — Syphilitic. Cachectic. Due to scarlatina, measles, lichen, prurigo, etc.

II.

GENERAL INFANTILE ATROPHY AND THE INJECTIONS OF SERUM.

The two pictures presented show very clearly the high degree of malnutrition present in severe cases of general infantile atrophy, and the marked efficacy of the subcutaneous injections of serum in such conditions.

Clinic Case. — On October 5, 1899, there entered my Clinic of Pediatrics at the University, a child that was so slender and had such a miserable appearance that he resembled more a mummy than a living being. We found him helpless on a bed, with half-bent limbs, somewhat rigid and apathetic; hair light and straight, skin of a dark yellow, dry, rough, and without any trace of subcutaneous adipose tissue. The skin on the inner side of his thighs hung in large folds. His greatly emaciated face, sunken cheeks with absence of "suction cushions," and a dark ugly color with deeply wrinkled lips, gave an ape-like appearance to the facies. The exhaustion was so great that he could scarcely raise a hand or even look around. Pinching scarcely provoked any response, and his suffering was made known by the increase in the wrinkles rather than by his cry. He was two years old,

This distrophy is peculiar to children under two years of age, and occurs with greater frequency during the first six months of life. Insufficient nourishment and diminished power of assimilation combine to produce the loss of weight and atrophy; *they* must be considered the principal factors in the etiology of this condition.

The causes of the essential atrophy are loss of nutritive power in the milk, in consequence of a reduction in amount or a diminution in the percentage of casein or fat or some of its nutritive elements. Moreover it may be caused by artificial feeding, food improperly administered, or mixed feeding prematurely instituted, and large losses of liquids caused by diarrhœa or fevers. Rickets, consumption, and other cachectic diseases cause secondary or symptomatic atrophy.

Infantile atrophy is essentially a disorder of a chemical character, intimately associated with the predominance of water in the immediate principles of the child, and depending on the general metamorphosis "which is very rapid in the young, because of both the rapidity of the vital processes, and the constant necessity of adding to the tissue of the body, besides keeping up the equilibrium. Therefore inanition is not tolerated for a long time." — Jacobi.[1]

If atrophy were not, as it is, a chemical perturbation with loss of the watery element of the tissues and the subcutaneous fat, we could not understand the promptness and the ease with which atrophy of the first degree is cured at times by a mere change in the diet, omitting mixed food, or regulating the nurse's regimen by prescribing a galactagogue, or, when other means fail, change of nurse. These rules being carried out, the scales will show that the child has increased in weight, and that this increase is progressive for several days, exceeding the ordinary record. Notwithstanding, it is sometimes necessary to use calomel or some other eupeptic to clear the digestive organs of anything difficult of assimilation in the bowels.

From what has been said, in my opinion *essential atrophy* should not be considered a septicemia, an acid intoxication, an auto-intoxication, an ordinary chronic gastro-enteritis, or a tabes-mesenterica. If some cases of atrophy have been fatal in consequence of furunculosis, with suppurating otitis and other manifestations of pus formation, in only a small number of cases have streptococcus and colibacillus been found. Although Czerny has seen respiration with deep inspirations and at long intervals, as in the coma of diabetes, this only happens shortly before death; the fresh organ extracts of children whose death has been caused by atrophy do not produce toxic phenomena in

[1] *Therapeutics of Infancy and Childhood*, p. 10.

animals. In nearly all cases of atrophy there is no fever, no persisting vomiting, no mucous diarrhœa, no intense intestinal pain. Nevertheless, this form of Marfan is sometimes accepted. Finally, it may be added, evidences of mesenteric tuberculosis are not found, and the disease is rapidly cured, as in the present case, with simple means.

We should recognize a mild form in which all the disorders are of a chemical nature with perhaps slight histological lesions in the intestinal epithelium. They are easily cured. There is also a severe form in which the general symptoms are very intense. The diminution of water in the tissues, the thickening of the blood, the difficulty in the circulation, the venous extravasations in some of the viscera, the shrinking of the brain, and the overlapping of the bones of the cranium may all cause serious complications and even death. In this form certain gastric and intestinal lesions of a specific character must be admitted.

In the autopsies performed by Baginsky one or two hours after death (in order to exclude the idea of any cadaveric lesions), he found the mucous lining of the intestine normal over large surfaces, and at the level of the Lieberkuhn glands he noticed a manifest cellular proliferation—some cells penetrated deep into the glands; in some places the outer layer of mucous membrane had fallen off, leaving these glands exposed. In brief, there was atrophy of the intestinal mucosa.

Fede has found a thinness of the mucosa of the stomach and intestine with dark or bluish spots, without any depression or ulceration. There was a congestion and infiltration both in the mucous and submucous coats, which were atrophied and contained small cells, the glands and cilia being constantly preserved, though somewhat shrunken through atrophy.

Treatment.— In atrophy, before treating the digestive tract, the blood must be attended to. Above all, it is necessary to consider the circulation, to increase the arterial tension, to prevent the thickening of the blood, the auto-intoxications, the venous extravasation in the viscera, the thrombosis and renal irritation, and therefore I agree with Jacobi's[1] idea, that in such cases "the remedy is water in sufficient quantities." Of the five paths that we may utilize, the venous, the arterial, the subcutaneous, the peritoneal, and the rectal, I consider the subcutaneous as preferable.

To accomplish the object, I have used for some years the subcutaneous injections of artificial serum and horse's serum in cases of atrophy. As the latter serum requires special facilities for its production

[1] *Loc. cit.*, p. 15.

and preservation, and as it has, moreover, a tendency to produce numerous varied skin eruptions, and as I do not consider its therapeutic virtues superior to the first, I have adopted as a more practical means the artificial serum. The solution of chloride of sodium, strength 10:1000, is employed in some cases, and solutions of sulphate of magnesia and of soda in others. In general, I inject in the abdominal wall 100 cubic centimetres, morning and afternoon.

I have begun to experiment with the serum from cow's milk, either pure or mixed with arsenic and other substances, but I have not yet gathered enough data to form an opinion as to its value.

Conclusion.— It is indispensable to include in the nosological tables *general infantile atrophy* in two forms: first, *slight*, curable; second, severe. This is accompanied by intestinal lesions and is apt to end with death. The most efficient treatment is the subcutaneous injections of serum.

(Kindly translated for Dr. Vargas by Mr. G. Picornell.)

III.

GANGRENA PULMONAR DIFUSA LATENTE.

El grabado adjunto representa un corte horizontal del pulmon izquierdo, en su posición normal, perteneciente á un niño que murió por gangrena difusa del pulmon.

He aqui la observación clínica: El dia 12 de Octubre de 1899 ingresó en la clínica de Pediatria de la Facultad de Medicina, un niño de tres años de edad, cuya respiración agitada, nos dió á entender desde el primer momento, que debia padecer de algun afecto respiratorio. Con efecto, mediante la percusión, pude apreciar que toda la zona correspondiente al pulmon izquierdo, daba un sonido apagado; esta falta de resonancia, se percibia hasta el espacio semilunar de Traube; en la parte externa y posterior del lado izquierdo, habia tambien falta de resonancia, por medio de la auscultacion, *comprobé* la ausencia de sonido en la respiración, de *ronchi* y de *crepitation ;* no habia tampoco vocal fremitus; en el pulmon derecho, encontré con la percusión una resonancia normal y con la auscultación, una respiración pueril, sin estertores ni ronquidos; el tubo digestivo estaba poco alterado. La temperatura era de 39°.5 ; las pulsaciones 142 ; las respiraciones 64 ; de cuando en cuando, habia tos, seca, insistente y ligera disnea.

Fuera de una lactancia defectuosa y de un raquitismo algo acentuado, que existia á la sazon, el niño no presentaba otros antecedentes patológicos, ni propios ni de sus padres; tan solo habia tenido un enfriamiento.

Diagnóstico—La primera impresión diagnóstica, llevábanos á aceptar ántes que una bronco-pneumonia ó una pneumonia, un derrame pleuri tico de poca intensidad; pero la falta de resonancia de la voz, la falta de egofonia y de resonancia expiratoria y el resultado negativo de la punción exploradora, me indujeron á desechar aquéllos diagnósticos y á aceptar el de *espleno-pneumonia*.

Prescribí al enfermito una gran revulsión en el costado izquierdo y en las piernas y una poción amoniacal.

Dia 13—Tuvo una pequeña hemoptisis; expulsó por la boca unos 40 gramos de sangre. Gran mejoria del estado general; Temperatura 37.7°; Respiraciones 60; Pulsaciones 136.

Dias 14 y 15—La tos fué mas blanda; la expectoración consistia en una saliva espumosa y blanca.

T. 37.6°C. R. 54. P. 120.

Le prescribí una poción alcalina y expectorante y embrocaciones con tintura de iodo en la pared torácica.

Dia 16—La macicez á la percusión fué menos intensa; en el espacio semilunar de Traube, el sonido era claro; por la auscultación se percibian estertores crepitantes y de grandes burbujas.

Dia 17—Sin causa apreciable, comenzó á subir la temperatura (38°.6) se presentó diarrea y la tos se puso seca.

Dia 18—Los signos físicos del pulmon no acusaban agravación del estado local, antes al contrario, revelaban disminución de la consolidación pulmonar y sin embargo, el estado general del niño, se presentó alarmante, por la palidez de la cara y mucosa labial, por la dilatación pupilar, por la debilidad cardiaca, por la agitación respiratoria y el marcado abatimiento: habia una gran *astenia* y esta resultaba inexplicable:, sin saber como, veíase venir la muerte, con cierta rapidez.

La *espleno-pneumonia*, era incapaz de darnos la explicación de aquélla gravedad, pues los sintomas locales, si acaso habrian revelado mejoria; un *derrame pleuritico abundante* que produjera dislocación cardiaca, era imposible de aceptarse en aquel caso, pues lo rechazaban la percusión y la auscultación y además resultaron negativas varias punciones exploradoras; tampoco cabia pensar en una auto-intoxicación de origen intestinal, toda vez que el tubo digestivo, aparecia casi normal; además *el aliento del enfermito era casi inodoro, no revelaba fetidez, ni fermentaciones gástricas ni de otra índole;* aquel niño moria cual si hubiera sido un *tuberculoso latente*, en el cual, el agotamiento producido por la toxina tuberculosa dominara toda la escena morbosa:, y sin embargo, los antecedentes que eran negativos, la falta de ganglios multiples infartados, la falta mas absoluta de los fenómenos respirato-

Latent Gangrene of the Lung.

rios precoces, todo en una palabra, rechazaba, en aquel niño la idea de la tuberculosis latente ú ostensible.

En los dias 19 y 20 se acentuaron estos fenómenos de debilidad general, y á pesar de usar los tónicos, el alcohol, la digital, la cafeina y las inyecciones subcutáneas de aceite alcanforado y de eucaliptol, *sin que ni los alumnos ni yo advirtiéramos olor de gangrena en el aire expirado*, murió el niño el dia 21 á media noche; esto es, á los 8 dias de haber comenzado la observación.

Autopsia—Levantada la pared anterior del torax, encontramos el pulmon izquierdo negrusco, desde el vértice á la base, con grandes oquedades llenas de un liquido gris, negrusco, con coágulos y bridas de tejido; despedia un olor *fétido y penetrante;* el olor propio de la *gangrena;* por su superficie, el tejido pulmonar estaba consolidado, carnificado, se hundia en el agua y estaba tan intimamente adherido á la pared torácica, que fué imposible separarle; por eso al levantar la pared anterior, se rasgó el pulmon dejando al descubierto la parte gangrenada; el corte transversal del dibujo dá una idea clara de lo extensa que era la destrucción pulmonar. Las investigaciones químicas é histológicas, pusieron fuera de duda que la lesión era *gangrenosa*. Examinados los ganglios y el resto del pulmon, no pudo encontrarse la más minima huella de tuberculosis.

Observaciones—Sin duda ninguna, este ha sido un caso de gangrena difusa del pulmon izquierdo, en el cual, por haberse unido intimamente la superficie á la pared torácica, no se ha producido una lesión pleuritica ni derrame ni pneumotorax, que lo habria puesto de relieve; y por no haberse abierto tampoco ningun foco en un bronquio, no ha podido presentarse la *fetidez del aliento* ni la *expetoración caracteristica* que habrian dado resuelto el diagnóstico.

Ha sido pues este, un caso de *gangrena latente* que sólo por la autopsia se ha podido diagnosticar.

Su realidad, constituye un proceso morbaso harto raro, que merece consignarse en la literatura por esta condición, y además, porque ofrece dos particularidades curiosas: una causa nueva y la hemoptisis prematura.

En la etiologia consignada por los diversos autores, no figura para nada entre las causas productoras, la espleno-pneumonia; es por lo tanto esta una nueva causa que debe figurar en los tratados; acaso podria asimilarse esta causa al infarto-pneumonia (infarction) consignado por Jacobi [1]; la *espleno-pneumonia* por el hecho de ser sus lesiones intersticiales, debia producir compresión de los capilares

[1] *Loc. cit.*, p. 497.

nutricios del pulmon y por consiguiente la lesión nutritiva que engendró el esfacelo; á pesar de que la hemoptisis hubiera podido disminuir algo la *compresión*, no fué bastante á impedir la gangrena; segun Rillietz y Barther, este sintoma de la hemoptisis, no es muy frecuente; en sus 26 casos, de ellos, 13 con tuberculosis, solo se registró la hemoptisis una vez por cada cuatro enfermos.

Entiendo que por efectos de la menor resistencia del niño, la gangrena del pulmon, máxime si es difusa, mata por auto-intoxicación, sin dar tiempo á que el proceso, por destrucción llegue á abrir una gran via bronquial ó pleuritica; en este caso la gangrena causa la muerte sin salir del *estado latente*, sin que se haya llegado á la *expectoración gris* ni á la *fetidez del aliento:*, por eso la gangrena latente del pulmon, es mas propia del niño que del adulto.

En resumen, paréceme que de este caso y de algun otro parecido puede establecerse la siguiente *regla clínica;* siempre que en el curso de un afecto pulmonar, con hemoptisis ó sin ella, y libre de tubercu losis, haya disparidad entre los sintomas locales y generales, esto es, que haya mejoria aparente del estado local del pulmon, al mismo tiempo que se agrave el estado general, por una astenia mas ó ménos intensa, será lógico suponer la existencia de una gangrena pulmonar latente, aun cuando falte el olor fétido del aliento.

ON ANTIPERISTALTIC MOVEMENT.

By GUSTAV LANGMANN, M.D.

INTESTINAL peristalsis is a complex process, the physiological explanation of which is still obscure. As commonly accepted the alternating circular constrictions and the rolling waves (Mall's irregular) are considered the means by which the food is pushed onward, the longitudinal swinging or pendulum movements (rhythmic of Mall) serve to churn and mix the food. The contact of the contents with the mucosa appears to be the principal stimulus for the muscular contractions. The question, however, arises: is peristalsis an automatic action of the smooth muscle-cells or is it caused by the ganglia which are found in extraordinary abundance in the plexus submucosus and more so in the plexus myentericus, or is it due to the direct influence of cerebro-spinal nerves? It would be out of place to enumerate here the many investigations in this line which have been repeated since the classical experiments of Van Braam-Houckgeest, and especially of Nothnagel, time and again (Gad, Luederitz, Mall, Pal), nor would it be expedient to enter into the contradictory results obtained concerning the innervation of the viscera. It may suffice to say that since Engelmann had attributed peristalsis to direct transfer from muscle-cell to muscle-cell, an opinion strengthened by the observation of the intestinal tract in lower animals, nearly all recent investigations firmly establish the paramount influence of the cerebro-spinal and sympathetic nerves. This view not only coincides with well-known clinical facts — phenomena of cerebral and psychical origin, — but also rests upon the experimental proofs that the splanchnic nerve acts as an inhibitor (Pflueger) and the vagus as an accelerator of intestinal motion.

While thus the definition of peristalsis is as yet far from completion, it is still more so with the so-called antiperistaltic movement. Let us define at the outset that by antiperistaltic movement we understand the actual reversal of the normal peristaltic wave, *i. e.*, a wave able to move contents from the rectum or cæcum towards the stomach, and not the "Rueckstosscontraktion" of Nothnagel (contraction of recoil),

a merely local contraction of the bowel just above an occlusion, which reaches but a few inches above its origin and is entirely ineffectual as regards the transportation of the contents. It is but natural that, the object of peristalsis being the gradual downward carriage of food, nearly all observers of our century have recognized only the onward peristaltic movement. Nevertheless, antiperistaltic motions were distinctly acknowledged and described by the best authorities of the eighteenth century, like Albrecht von Haller and Morgagni, a fact almost forgotten, or at least ignored, by modern physiologists and clinicians. It was Engelmann also who in 1870 pronounced the regular occurrence of an antiperistaltic wave, coinciding with a peristaltic one upon stimulating the serous surface of any part of the bowel. Having established this phenomenon previously in the ureter, he formulated the law, that upon stimulation of smooth muscle fibres a peristaltic wave runs off in both directions. The bowel in these experiments was exposed to cool air, but after Braam Houckgeest's initiation, all investigators worked in a tepid bath of normal saline solution (or under similar conditions). Thus Nothnagel found that Engelmann's law does not hold good in experiments performed in salt solution. Pal confirms this rule in rabbits, but he finds in dogs and men no difference as to the effect of air and salt water.

One clinical symptom, however, of frequent occurrence seems to necessitate the assumption of antiperistalsis: the stercoral vomiting of ileus consequent to intestinal occlusion. After lively discussion during the past years, the old theory of Van Swieten, or rather Haguenot (1713), advocated also by Morgagni, has come to the fore again, viz., that the stercoral vomiting is nothing but the overflow of the fecaloid masses which, gradually filling the intestine above the obstruction up to the pylorus, are expelled through the mouth by the vomitory action of abdominal pressure.[1] One point is to be specially emphasized and that is, that the matter ejected in ileus is liquid or soft; cases in which solid feces were reported to have been vomited, are doubted by the best authorities as being incorrectly observed. It has often been demonstrated since Morgagni's and de Haën's time, that the contents of the ileum may resemble exactly normal feces of the colon. Moreover, Hermann, by isolating a coil of ileum, proved that even the inspissated intestinal juice forms hard fecal masses without the access of food or bile.[2] Nevertheless, Leichtenstern, in his admirable report on ileus,

[1] French writers always differentiate between "matière fécaloide and matière fécale, fèces moulées" (formed feces), in German writings "faeculent and faecal." It would be advisable to adopt the same distinction in our writings.

[2] It may be of interest to mention here a few instances of fecaloid vomiting in cases of

advocated the possibility of antiperistalsis in those cases where a sudden strangulation or twisting of the bowel induces vomiting of fecaloid masses, by reflex irritation from the peritoneal surface, before the intestinal canal could have filled up. Kirstein's experiments are still more instructive. He divided the small intestine of a dog near the ileo-cæcal valve and closed the ends by aseptic suture. The dog died after six weeks, and never vomited stercoral nor any other matter at all. He found an enormously dilated cylindrical lower end of the ileum, filled with soft greenish feces. On the other hand, dogs with a tight ligature of the lower ileum ejected fecal masses, while the parts above the ligature were perfectly empty and immobile. The first experiment proves that complete occlusion of the bowel and the mere accumulation of feces are not sufficient to cause vomiting, while the second experiment shows that injury of the serous surface of the intestine brings on by reflex irritation a disturbance of the motor functions of portions of the gut which are far distant from the incarcerated coil.[1] In fact, the entire theory of dynamic ileus, the "paralysie par réflexe," *e. g.*, when an incarcerated omentum or uterus, a twisting of an ovarian tumor, brings on a most violent attack of ileus, militates against Haguenot's doctrine. It is proper perhaps in this place to call attention to the different effect upon the intestinal motion, when the faradic current is applied to the serous or to the mucous surface of the bowel (Pohl, Meltzer).

Until recently the only uncontested instance of antiperistaltic movement was Nothnagel's application of crystals of sodium salts to the outer and inner surface of the intestine. This movement may run as far as 8 cm. upward from the point of contact. In one instance, when a strong solution of salt was injected into the rectum of a patient on whom laparotomy was performed, it rose to 45 cm. above the ileo-cæcal valve. Luederitz produced a similar effect by distending the bowel with an inflated rubber bag. Then in 1884 Gruetzner reported that, after injecting small particles like lycopodium, coal, or short clippings of horse hair, suspended in normal saline solution, into the rectum of rabbits, guinea-pigs, and rats, he found them a few hours later in the stomach and almost none in the intervening parts of the intestines. These experiments have been repeated by many investigators (Chris-

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cancer of the pylorus (Boussumier, Schreiber). They are easily explained: the pylorus is both stenosed and kept open by the rigidity of the carcinomatous wall; the retention of food in the stomach incites vomiting spells which, by abdominal pressure, force the contents of the duodenum and ileum back into the stomach.

[1] Nothnagel's experiments on this point, which do not agree with Kirstein's, were performed exclusively on rabbits.

tomanos, Dauber). Most of the results were negative, and Gruetzner's successes were attributed to the imperfect muzzling of his animals, which licked up some of their own feces. Several observers, however, confirm Gruetzner's assertions; and Gruetzner himself, after a new series of experiments, carried out with all precautions, still clings to his first conclusion, *i. e.*, that antiperistalsis is an approved fact. Riegel may be correct in saying that the epithelium of the intestinal mucosa is responsible for the transportation of those small particles. But why should it act in these cases in a reverse direction? At any rate, it is well conceivable, that a strong salt solution of 20 or 30% may create osmotic currents which on the other part generate a countercurrent, but why a solution, having the same salinity as the surrounding tissue, should stimulate antiperistaltic waves, is not evident. Mall, in speaking of the irregular rapid wave which may pass through the whole length of the intestine in one minute and which runs in both directions from any point of irritation, finds it also pathological, as does Nothnagel, since it is observed in a gut rendered more irritable through anæmia or hyperæmia or by impaired digestion. Under these conditions he finds complete antiperistalsis from the rectum to the stomach intelligible. Several experiments, made by excising a part of the ileum and sewing the ends in a reverse direction, demonstrate that the peristaltic wave in the excised loop is not changed thereby (Kirstein, Ballance, and Mall). It was found that food passes to a certain extent through the reversed part, but that the upper suture was considerably dilated by the opposing action of both peristaltic waves. Moreover, Mall saw that after sixty-three days the reversed loop reacted to the induced current in the same direction as it had done before the operation. This point wants further elucidation by experiment.

Physiological experiments, then, have as yet not conclusively proven the occurrence of antiperistaltic waves. There are, however, clinical instances which will not admit of any other explanation but that of antiperistalsis. They might be called proofs of a recommendation made by Gruetzner in his experiments on antiperistaltic movements, viz.: to add salt to all nutrient enemata, in order to carry them up into the ileum for more complete digestion. It is but fair to state that this same idea was advocated in 1878 by H. T. Campbell, in an elaborate paper before the American Gynæcological Society. He based his recommendation on those facts alluded to above, *i.e.*, the not unfrequent report of nutrient enemata having been vomited. Woodward quotes several cases of good unbiased observers (Hearn, Battey, and Copeland) who saw clysters of beef tea, soap water and of castor oil thrown up by the mouth. Ficklen's patient objected to the continuance of enemata

of beef tea, because she invariably tasted it in the matter vomited. Harris's patient after ovariotomy vomited enemata of beef tea. Frank's patient, a travelling agent of nineteen years, debilitated by pleuro-pneumonia, vomited enemata of beef tea two or three times a day for nine days. Constipation and vomiting spells had preceded these occurrences. Routh says "that persons who had taken cod-liver oil by injection complained to him of feeling the taste of it in their mouth for hours later. This is equally true as regards children."

These cases call to our mind the violent opposition formerly made to such observations because of the supposed impermeability of the ileo-cæcal valve to a retrograde current. Almost from the very time of Morgagni's classical description of the valve (Adversaria anatomica) it has been shown that, at least in the majority of the cases experimented on, the valve was not impermeable to fluids injected into the rectum. Thus de Haën succeeded in "pumping many pounds of water through his dogs," and, strangely enough, while the stomach was found to be well filled, the intestines were empty. This procedure has been repeated many times since, partly for experimental, partly for therapeutic purposes. Let us recall only Cantani's enteroclysis, recommended and practised in cholera. Genersich relates that it was much easier to fill the whole intestinal canal from below than from the mouth. An enteroclysis is, however, not possible in all cases; it depends upon the configuration of the valve. When the upper lip overlaps or both lips are equal, the valve is water-tight; with a shorter lower lip, a current from the cæcum passes into the ileum. Debierre found that 60% of the cases investigated presented this latter type.

A still stronger opposition has always existed towards a group of clinical observations which are collected under the convenient name of *ileus nervosus*, *i. e.*, stercoral vomiting without mechanical obstruction or incarceration of the bowel. Such cases have been on record for centuries, and the older they are, the odder they sound: the ordinary fecal emesis is associated with some of the most marvellous tales, wherein we hear of medicines, suppositories or candles having been swallowed up by the rectum and ejected by the mouth. No wonder, therefore, that the whole subject was discredited by the sensible part of the profession. Morgagni, who cites a large number of these reports, disputes energetically their validity, although he admits antiperistaltic movement in his principal work (*De Sedibus*, etc.). Even to-day this topic is regarded with suspicion by physicians in general. The mildest criticism allows that they are based either upon careless observation, or are the result of clever deceptions on the part of the patients. It seems indeed that most observers of former years have been rather

reluctant to publish their cases in order to avoid the charge of inaccurate observation or even a flat contradiction. The publications of the very last years have gradually shown more interest and belief in these forms of disease. There are still but few well authenticated cases. They are scattered through the literature of all countries and, though quoted in special treatises, are mostly hidden in journals and little accessible to the general reader. In connection with one case of my own, it is the object of this paper to refer to as many reliable cases as possible, in order to present this interesting subject collectively and to draw some conclusions, both in regard to antiperistaltic movement and to some eminently practical points in diagnosis.

In continuance of what was said above about the distinction between fecaloid and stercoral masses, it ought to be stated that it is characteristic in most of the following cases, that hard-formed feces were ejected by the mouth.

Let us begin with my own observation:

N. S., twenty-one years, teacher, entered the German Hospital of New York, April 18, 1889. Father died of kidney, mother of unknown disease. Had pneumonia in her eighth and eighteenth years. Inflammatory rheumatism in summer 1888, after which palpitation. Menstruation commenced at fourteen years, stopped between seventeen and eighteen, since then regular without pain. Four years ago fell from a staircase and broke neck of left femur. Was treated for a long time by extension (left leg paralyzed for nine months?). Since that time occasionally severe pain along the spine. Vomited much in fall 1887, even blood. From February to April, 1888, perito tis on left side (while in Paris); vomited then blood and feces; after that constipation and often cardialgia with emesis. December 1888, again peritonitis on left side and hematemesis. Since the beginning of March more vomiting, often blood. Since a week only able to take gruel, which is thrown up also. During the last month has habituated herself to hypodermics of morphine, sometimes two or three times a day.

Status præsens: Well developed, well nourished; appearance not in accord with the tale of her suffering. Lungs normal. Slight systolic murmur at apex of heart. No meteorism. Left hypochondrium sensitive, especially at one fixed point, also the left side of abdomen down to groin. No other signs of hysteria present. Spine very tender upon pressure from third vertebra to sacrum; painful between 11th and 12th dorsal vertebra. Urine acid, 1030. No albumen. Sexual organs normal, hymen intact.

April 18th. All food thrown up. Iced milk, bismuth, morphine hypodermically. Supposit. opii.

April 19th. Temperature 99° (normal all the time). Vomited everything, therefore nutrient enema.

April 20th. About fifteen to thirty minutes after enema, ejects fecaloid liquid. Hyoscyamine 0.0003 grm hypodermically twice a day.

April 23d. No feces vomited. Irrigation of stomach and feeding by tube with milk and bread.

April 25th. No vomiting. Faradization of spine.

April 30th. Feeding by tube three times a day.

May 3d. Tinctura asæfœtidæ, three times per os.

May 10th. Vomiting spells return. Asafœtida stopped.

May 10th. Vomits fecaloid liquid. Asafœtida per rectum.

May 18th. Throws up liquid and hard feces.

This stercoraceous vomiting was repeated now several times; hard scybala, an inch thick, about 50 c. c. at one time, were thrown up. A fistulous communication between the stomach and colon seemed most likely, after the repeated hematemesis and the circumscribed pain in the gastric region. To make sure of this, a mixture of two grammes of indigo with 200 c. c. of tepid water was injected into the rectum. After less than fifteen minutes, while I was talking to the patient at an adjoining bed, feces mixed with indigo were thrown up by the mouth. This occurrence was so convincing that other means, like inflation of the rectum, were thought superfluous. If I had known at that time the experience of Briquet, related further on, I would have been more careful. In a consultation with Dr. Willy Meyer, it was decided to perform laparotomy and sew up the fistula on either side. The patient consented to the operation most cheerfully. On June 1st, Dr. Willy Meyer opened the abdomen by a median incision; stomach and colon were found not adherent and apparently healthy, except for a darning needle of about four inches which was impacted in the anterior wall of the stomach, lengthwise between serosa and mucosa, apparently at the spot where the most severe pain was felt. The wound was closed and healed per primam, with the exception of the upper part where the patient herself had disturbed the dressing. This healed well after a secondary suture.

The emesis had ceased entirely. On July 3d, however, nausea with vomiting of mucus and blood commenced again. Enemata did not excite stercoraceous vomiting, but on July 10th feces were thrown up spontaneously, and for three days after liquid and hard feces together; sometimes more than 50 c.c. were ejected. It seemed to be a favorable time now to experiment in regard to the carriage of substances from the rectum to the mouth. Accordingly, on the forenoon of July 15th the stomach was washed out. Nothing but some coagulated milk was emptied. All cups, basins, towels, and handkerchiefs in reach of the patient were removed, except a glass basin for possible vomiting. Then the patient was placed on her right side, and at 11:01 A.M. two grammes of indigo, suspended in 200 c. c. of tepid water, were injected into the rectum. The anus was cleaned and a white towel spread under it. The patient remained in this position with her hands on the outside of the blanket and was watched by myself, my assistant Dr. F. Zitz, and the head nurse who, while apparently occupied in the neighborhood, kept a constant eye on the patient. At 11:10, exactly nine minutes after the introduction of the enema, a feculent mixture of milk coagula with much indigo was vomited into the glass basin. Anus and hands of the patient proved to be perfectly clean. Bluish matter was thrown up several times during the day, and blue feces also took the natural course. Two days later a mixture of alkanet was injected under the same precautions. After an hour's watching, no vomiting. Some masses ejected a few hours later were, to my regret, thrown away unobserved. July 19th, I made another injection of indigo which excited no vomiting. In fact, after this, emesis stopped spontaneously. July 22d, the head nurse reported that some hard feces, wrapped in paper, were found under the patient's pillow. The news spread at once through the ward, and under the overwhelming impression of being detected, she requested to be discharged. Not to lose an interesting case so suddenly, I persuaded her to stay, assuring her that she was not responsible for her acts. The taunts of the other inmates, however, caused her to leave on July 29th. She later tried different tricks at another hospital and at a downtown dispensary, but after being again detected she was lost sight of.

Undoubtedly one objection will be made to this report: that we were the victims of a clever deceiver, as proven by her attempt to collect her feces for occasional future use. I leave it to the reader to decide whether deception was possible under the precautions described

above. My belief is that the stercoraceous vomiting was genuine and spontaneous, that the patient was unable to bring it on at will, and only after perceiving on what point the medical interest centred did she try purposely to repeat a symptom which in her altered condition she was unable to bring on.

The following cases have been selected as best illustrating the disease in question :

Briquet : Very often cited in special treatises, but very little known in extenso. It is so characteristic that it is worth while to repeat part of it with Briquet's own words : "A girl of twenty-seven years entered the hospital in May, 1857, to be treated for various hysterical symptoms, the most troublesome of which was a continuous somnolence. We meant to rouse her by giving coffee, but after a few days she could not tolerate this infusion, the abhorrence of which caused her to vomit immediately after drinking. Therefore an infusion of 32 grammes of coffee and 600 c.c. was administered by enema. This injection, taken with extreme aversion, soon caused nausea, colic, rumbling, then retching, and after half an hour a liquid was rejected by the mouth, having exactly the color and odor of coffee. The quantity ejected was estimated to be about one third of the liquid given by enema. To avoid all error, after the elapse of two days, we gave her 500 c. c. of a decoction of coffee by enema. This was done in my presence during the morning visit, and no sooner was it given than the same occurrence took place as before. After all sorts of ill feeling, the decoction was thrown up at the end of fifteen minutes, without being mixed with any other substance. During all the time between the injection and the vomiting, the patient did not leave her bed and was constantly watched, so that she never could have taken any coffee concealed somewhere under her bed. After several repetitions of the same experiment, to dispel all doubt we took a substance never used in the household : we injected tincture of litmus at the very minute when it was brought in from the druggist's. This substance had never been in the ward. The patient imagined she was getting coffee, but not more than twelve minutes after the enema, the tincture of litmus was vomited, its blue color turning red. Finally an injection of salt water was given, and fifteen minutes later the patient vomited a salty liquid which, treated with nitrate of silver, showed abundant precipitate of chloride of silver."

Jaccoud : "In 1867, a young woman with hysterical convulsions was admitted to the hospital. Two weeks later she became completely constipated and, without notable meteorism, commenced to vomit stercoral masses, not those fecaloid masses of common intestinal obstruction, but veritable solid, cylindrical excrements of brown color and characteristic odor. At a glance one could see that they came from the large intestine. A secret watch was placed around her, but on the third or fourth day one of these vomiting spells came off during the morning visit. The masses thrown up were like those described. In fact it was a complete defecation by the mouth. The patient, feeling well otherwise, stopped vomiting on the eighth day, and the feces took their usual course. Ten days later she was taken sick with typhoid fever, and after her death, which took place in the third week, except the lesions of typhoid absolutely nothing was found in the intestines. The ileo-cæcal valve showed its usual dimensions."

Jaccoud soon thereafter met another hysterical woman who presented for several days the usual signs of intestinal obstruction with fecaloid vomiting. When preparations for an operation were made, an antispasmodic treatment re-established normal defecation.

Rosenstein : A boy of nine years exhibits convulsive twitching from February 1st to March 7th, together with passage of stercoral masses through both mouth and anus. Scybala 2 to 3 cm. long and 5 to 8 cm. thick, once a mass of 18 cm. in length, were thrown up. Enemata with alkanet gave a blue color to both stool and vomit. Following tetanic

stretching of the arms, then trismus, opisthotonus, oppressive feeling about the chest; two or three condensed scybala were passed out of the mouth. No peristaltic movements visible through the well-nourished abdominal wall. Boy was cured by bromides.

De Tullio: A married woman of twenty-nine years, who had been subject to hysterical convulsions and abdominal pain, with emesis of large quantities of water, previous to her marriage, was again attacked some years later, following a strong mental emotion. Unilateral anæsthesia and hyperæsthesia, globus and convulsions, also uncontrollable vomiting. Uterus in retroflexion fixed to the rectum by firm adhesions. For a constipation of five days' duration a clyster of two liters of soap water was given which, after some uneasiness in the stomach, was ejected out of the mouth fifteen minutes later. This performance was repeated often, from November to March, 1887. Of the greatest interest is the observation of the mechanism of antiperistalsis. "In the moments of greatest suffering" says De Tullio, "mostly just preceding a violent fit, one could perceive the intestinal movements on the outside of the abdomen. Starting from the sigmoid flexure, they rolled gradually up the descending, transverse, and ascending colon, then the mass of the small intestine, to close as if in a convolution at the pylorus, where a slowly enlarging tumor was well to be seen until the pyloric valve was forced and emesis took place. These movements increased rapidly with the clyster until the injected fluid was vomited; then followed a relative calm of the intestines, which were in a continuous gentle motion." The vomiting spells were always stimulated by the introduction of some substance into the stomach or rectum. Never were fecal masses thrown up. All symptoms disappeared after the operation for retroflexion.

De Tullio: Young girl of fifteen years showed signs of psychical emotions for some time. After a short fever she developed hysteria with convulsions and meteorism; stercoral vomiting repeated for about two weeks after clyster of oil. The enemata, watched with great care, were ejected by the mouth about an hour after the administration, mixed with dry feces, sometimes as large as a hen's egg. By rectum fecal masses, compressed and tape-like, were sometimes passed.

Cherchevsky: This case often quoted in French literature seems doubtful. An educated neurasthenic, high official, forty years of age, after a constipation of ten days, tries by hard pressing to bring on a passage. Instead, a yellow piece of excrement, 8 cm. long and as thick as a finger, is thrown out of the mouth. An hour later a copious stool is effected.

Fouquet: A seamstress of twenty years, hysterical, is seized in the street with violent colic, which is followed by vomiting, first of food, then of hard excrements. This latter vomiting is repeated several times. A constipation of twenty days preceded this attack. A similar accident occurred three months previous. Patient developed other symptoms of hysteria and succumbed a year later to phthisis.

Cullen: "We had a patient in the infirmary, who for weeks threw up stercoraceous substances and the matter injected by glysters. There was an entire absence of fever, and the disease by its circumstances and cure shewed that no inflammation was present."

Voisin: A young hysterical girl, in whom symptoms of occlusion developed three times, was saved twice by antispasmodics and purgatives; the third time she succumbed. The autopsy revealed the traces of a purely spastic contraction of the bowel.

Campbell: In speaking of the value of rectal alimentation quotes a patient of Dr. Harris, who was operated on for ovarian tumor by Dr. Atlee, of Philadelphia. Excessive vomiting supervened; beef tea given by enema did not escape per anum, but was vomited from the mouth. Stercoraceous masses preceded the beef tea. Dr. Atlee, being consulted about this feature, replied that he had observed a similar occurrence from inverted peristalsis in one or more of his former ovariotomies.

Desnos: Young hysterical man who is subject to epileptiform fits, throws up formed

feces, either during attacks or in the interval. He claimed to have had stool exclusively by the mouth for two years.

Bryant: Fourth laparotomy performed on a female artist of twenty-three years. She claimed to have had inflammation of the bowel on the left side three times. Laparotomy had been performed three times. She vomited blood and passed bloody stools. Nutrient enemata were thrown up by the mouth. A communication between stomach and colon being suspected, an enema of glycerine with methylene violet was administered, and was vomited in fifteen or twenty seconds. Laparotomy on February 27, 1892, revealed adhesions between transverse colon and stomach, yet no communication. An enema of pyoktanine, injected during the operation, was seen in the colon, but did not rise up, Further deceptions with temperature were detected, and finally patient admitted fraud in vomiting also.

Mikulicz: To warn against the deceit on the part of hystericals who crave for operations, reports a case of a woman who exhibited hematemesis and stercoraceous vomiting. From her forty-eighth to fifty-first year she underwent five different operations mostly for the supposed ileus. Just previous to a sixth operation she was caught in deceiving.

Leube: Girl of nineteen years, with nervous vomiting and normal abdomen, suddenly vomited stercoral matter. The whole length of the colon, from cæcum to sigmoid flexure, could be felt as a hard cord as thick as a finger, only the rectum being free. Twenty-four hours later this symptom had disappeared; no meteorism. After ten days the same symptoms presented themselves without inflation of the bowels. Patient cured.

Strauss: Man of twenty-nine years with distinct traumatic hysteria suffered with immense meteorism, obstinate constipation, and fecal vomiting. Laparotomy was performed twice for suspected intestinal stenosis. Meteorism disappeared suddenly and spontaneous stools set in.

Treves: Woman of twenty-four years had several operations performed on her abdomen for pain. Five months previous to admission to hospital a constipation of four weeks with fecal vomiting; the latter was believed to be due to gastro-colic fistula. A few weeks after admission bowels ceased to act; nutrient enemata were thrown up by the mouth, also fecal masses. (Temperature in mouth rose to 109°.) She was watched and castor oil injected into the rectum. In ten minutes it was returned by the mouth. Next a pint of water, colored with methylene blue, was given. In ten minutes exactly, a pint of water of a blue color was rejected by the mouth. As she grew worse, Treves reopened her abdomen and found the colon normal. After the operation she went through the performance of dying, but after isolation all symptoms ceased with the absence of an appreciative audience. "She explained how she managed to raise the temperature, but it was never ascertained how she managed to bring the enemata out of her mouth."

Murphy: Operated on a man of forty years who previously had had several attacks of lead colic, but now after five days of constipation showed symptoms of obstruction. After a median incision an enlarged coil of the intestine presented itself. By this were pulled out eight inches of a contracted portion which resembled a solid cord, three eighths of an inch in diameter and as stiff as a rope. The intestine above was distended to two and a half inches in diameter. After ten minutes' exposure to the air the spasm began to subside in the proximal end; after twenty minutes the bowel had expanded to about one inch diameter, when it was returned and the wound closed. Three hours later the bowels moved of themselves.

Woelfler: Girl of thirty-one years had for a long time abdominal pain and vomiting spells, anuria, finally stercoraceous vomiting. At the operation no mechanical obstruction was found. Large intestine filled with hard scybala, small intestine at several places tightly contracted between two hard scybala. Wound closed, broke open again after six months, when hard feces passed out there. Anus præternaturalis was closed four years later.

Slajmer: Nun of twenty-six years, very hysterical, frequent abdominal pain, chiefly in left side. Laparotomy after a constipation of eight days and stercoraceous vomiting.

Intestinal coils inflated; about two metres below pylorus a portion of the ileum of 20 to 25 cm. contracted like a ribbon: bowel below this spot normal, above it enlarged to its triple size. When the bowel was taken out the contraction relaxed, but the marks of constriction remained distinctly visible. Spontaneous evacuation of the bowel the following day. Intestinal symptoms, which appeared again later on, were subdued by antispasmodic treatment.

Schloffer: Married woman of twenty-eight years, treated a long while for hysteria, hematuria, peritonitis, etc.; after a constipation of ten days began to vomit pieces of excrements as much perhaps as a copious stool. Laparotomy performed immediately. Eventration of the entire small intestine revealed nothing but a firmly contracted piece of the lower ileum about 10 cm. in length; at several places small scybala could be felt; large intestine soft, of normal dimension. After closing of the wound the patient was told that the operation was successful in removing the obstacle. She made a good recovery.

A number of the cases recorded above have a somewhat fantastic appearance which, though resting on the authority of most reliable observers, might not appeal to the credence of the sceptical. In some, trickery alternates with reality; in others it seems to dominate the whole disease; and in others again even the attending physicians believe themselves throughout the victims of clever deceit. It appears to me that, even when fraud was detected, the patients have had at some previous time a genuine emesis of fecal masses; such an accident, unknown to them and unheard of, strikes them at once as an extremely interesting symptom, and only when nature fails to exhibit it spontaneously, do they resort to fraud. The experienced Gilles de la Tourette, in deprecating the general tendency to attribute hysterical symptoms to simulation, claims that Bryant's case, though Bryant himself believed to have been deceived throughout, is nevertheless genuine; and the patient of Treves, who evidently supposes the rapid appearance of the colored enemata at the mouth to be a proof of fraud, is an exact counterpart of my own patient.

I may refer here to a test made by myself in relation to the possibility of violent emesis to bring on stercoral vomiting. I am indebted for these experiments to Dr. M. Rosenthal, at that time house physician of the Montefiore Home.

Fanny G., twenty-one years, for five years hysterical convulsions and occasional vomiting, during the last year ischuria, hematuria, no nephritis; six months ago suprapubic cystotomy in a hospital, after that incontinence. Claims to have vomited everything during the last three months. No constipation. Nutrient enemata not vomited. In March, 1890, her stomach was washed out every forenoon and thereafter an enema with indigo or alkanet was administered (five times). The matter vomited during the day did not show any trace of color, but several masses, said to have been ejected during the night, did contain some of the dyestuffs. These tests were rejected as possibly fraudulent; the experiments, in this case at least, prove that emesis alone, no matter how violent, will not cause stercoral vomiting.

Some people may insist that no hard feces are able to pass the ileo-cæcal valve and that their appearance in vomited matter is evidence

of fraudulent admixture. The cases related by Schloffer prove that masses may form in a constricted ileum and, no matter how copious and hard a stool may appear, it does not necessarily take its origin in the colon. In the perusal of these cases it is seen that fecal emesis is usually preceded by prolonged constipation. The feces in the ileum have had sufficient time to become inspissated and to assume the appearance of contents of the colon. When we see liquids pass all the way from the rectum to the mouth, we may easily be misled to assume, that solid feces mixed with liquid may travel the same distance. The greater possibility, however, is that they are carried along from some deposits in the ileum. Yet it is conceivable, that an ileo-cæcal valve may be forced by a gaseous tumor which, when preceding a wave of the intestinal contents, may open the passage wide enough for solid matter.

Constipation is not the only abdominal ailment of which these patients complain. The latter invariably belong to that extensive class of neurasthenics and hystericals who fill every day the ear of the patient practitioner with the laments of—as we may wrongly suppose —imaginary or exaggerated sufferings. Colicky pains in various places, diarrhœa, constipation, tumors, membranous discharges, hematemesis and ileus may be the different symptoms which one and the same individual may present to us in the course of time. Our handbooks are compelled, while anatomical researches are still lacking, to set up an artificial classification, but nothing prevents our considering all the different ailments enumerated above as the manifestations of the same cause, varying but in degree. Enterospasm is one of the most generally acknowledged forms of intestinal neurosis, and obstinate constipation may be directly referred to it, as is shown by the last of our reported cases. How many times may colic, meteorism and transient constipation have preceded, until finally a constriction, either limited to a few centimetres or extending throughout the whole length of the colon, lasts long enough to obstruct all passage downward! In almost all instances the obstruction was removed by spontaneous relaxation, but it ought not to be forgotten, that at least in one case (Voisin) the spasm lasted long enough to bring on fatal exitus.

To the same cause may also be attributed the other perverse action of the intestinal nerves, the antiperistaltic wave. There is no perversity of nerve function which we are not prepared to meet some day in a hysterical person. The oldest and most frequently encountered symptom of hysteria is the ascending globus, the sensation of a ball rising not only from the stomach but even from the hypogastrium to the throat (Jolly, Loewenfeld). Similar sensations are described in

male sexual neurasthenics (Peyer). We have the testimony of De Tullio's case, how nature may produce this sensation and how in every case an actual, though invisible, wave may rise. And while in some instances it may create nothing but a sensation, in others it may be strong enough to carry substances along. Doubtless there can be no reasonable objection to the supposition of an antiperistalsis; the often attested rising of enemata cannot be explained in any other way.

The rapidity with which the enemata are carried to the mouth, suggests also an active co-operation of the muscular coat of the intestines. In Gruetzner's experiment it took hours to carry small particles like coal and lycopodium to the stomach. Mall figures that the normal peristaltic wave must take at least an hour and a half to move a certain object from the pylorus to the cæcum; the irregular rolling wave, which is considered by Nothnagel to border upon the pathological, runs the same distance in less than a minute. It is waves of this latter rapid sort which we must expect to find acting in antiperistalsis. Evidently there is also a difference among these phenomena in different patients. De Tullio in his observation does not state the time of the visible antiperistalsis; it may be supposed to be the same as in the rejection of an enema, *i. e.*, fifteen minutes. If, therefore, we take antiperistalsis for granted in those cases where there is no actual obstruction, it is but one step to admit it also in spastic constriction. Morgagni in his collection of cases (*De Sedibus*, etc.) considers the shortness of time which elapses between the enema and the attack of vomiting (one hour or a quarter of an hour) as conclusive evidence of careless observation. We are doubtless entitled to accept some of the statements as genuine.

In closing, then, it may be said: there exists a true antiperistalsis as a pathological phenomenon, chiefly in intestinal neurosis. It sometimes runs off with such extraordinary rapidity, that it may give rise to grave mistakes in diagnosis.

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TOTAL EXTIRPATION OF THE URETER.

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TOTAL extirpation of the ureter means the removal of the entire canal from a point just below the pelvis of the kidney to its very entrance into the vesical wall. The operation is done either simultaneously with nephrectomy (primary) or some time, sooner or later, after this interference (secondary). The primary operation is rarely carried out. It means for the patient a serious addition to the risk connected with removal of the kidney; it requires more time and, consequently, a longer general anæsthesia, both important factors in this particular operation; in cases of suppurating or of tuberculous kidney it will often render impossible the task of maintaining an aseptic condition of the large retroperitoneal wound. Tamponade followed by secondary suture will then become necessary. This complicates the after-treatment and prolongs convalescence. We shall, therefore, oftener hear of secondary than of primary total ureterectomy.

Inasmuch as the operation is always simultaneous with or consecutive to nephrectomy, the same pathologic condition that had induced the surgeon to extirpate the kidney will be found to exist in the ureter.

Those who have followed the evolution of renal surgery know that conservatism has taken a firm hold in this chapter of our science. To-day the kidney is at the first operation removed only in cases of tumor, primary tuberculosis, and exceptionally severe cases of suppuration; and even in these affections a few authors have recently argued against sacrificing the entire organ. Whether such an attitude is justified, the future will show.

It appears proper to review separately these three indications for primary nephrectomy recognized to-day by the majority of surgeons, with regard to their influence upon a probably following total extirpation of the ureter.

1. *Tumor of the Kidney.* As a matter of course, only malignant growths come into consideration. Of these I exclude such as did not primarily originate in the kidney. As far as I have been able to ascertain, a case of total ureterectomy for carcinoma or sarcoma of the kidney has not been reported in literature. Of course, if the ureter should appear to be involved in the disease, no surgeon would hesitate nowadays to make his operation a radical one, even if he should be obliged, owing to the serious condition of his patient at the time of the first operation, to wait a few days before doing ureterectomy. Nevertheless, it is evident that, in the majority of cases, the ureter need not be removed entire. If the latter organ has become involved in the disease to its entire length, the trouble will in almost every instance be beyond the reach of the knife. Thus, ureterectomy in cases of malignant renal tumor will generally be a partial one. The operator will do wise, in every instance of this kind, to remove as much of the upper portion of the ureter as he can reach from the wound, without subjecting the patient to an additional operation. If he has done that, he will have carried out what appears to be necessary in the given case.

2. *Tuberculosis of the Kidney.* In fourteen cases of renal tuberculosis where I had to perform nephrectomy, the ureter was not totally extirpated. In all these the wound closed without leaving a sinus, although scraping and proper treatment of the wound became necessary, now and then, towards the end of convalescence.[1] If materially infiltrated, the proximal part of the ureter was resected as far down as possible; if not especially affected by the tuberculous process, the tube was simply cut off below the kidney and dropped back into the retroperitoneal space. The latter procedure I practised up to several years ago. However, since I have observed a case in which the greater part of the urine from the healthy right kidney was discharged through a fistula in the left loin where the ureter had been simply cut off after extirpation of a tuberculous kidney, I have modified my method, and now invariably cauterize the lumen of the distal end with a Paquelin, and then tie it with catgut. Prior to operation, the cystoscope had shown, in the case just referred to, the mouth of the right ureter to be healthy; that of the left, much ulcerated. By the ulcerative process the physiologic valve-action of the ureteral mouth had been completely destroyed. The urine, after having descended from the right kidney, simply ran up and out of the open lumen in the left lumbar region, principally when the patient wsa in a recumbent posture.

[1] In one case, I learned lately, the lumbar wound broke open again about eight months after nephrectomy and necessitated the removal of the upper portion of the ureter.

Primary total ureterectomy for tuberculosis is certainly rarely indicated. Howard A. Kelly, of Baltimore, James Israel, of Berlin, and a few others have reported such cases. Here it was seen during nephrectomy that the ureter was very seriously affected. Partial resection of the renal portion would have been equivalent to an incomplete operation.

In the majority of this class of cases, however, I think, the ureter will take care of itself, if the case had been correctly diagnosticated in the beginning of the disease, and then promptly subjected to nephrectomy. With the source of the trouble, viz., the primarily diseased tuberculous kidney, removed, the continued re-infection of the ureter ceases, and the tuberculous process within the ureter heals. The cystoscope nicely corroborates this assumption. In the majority of cases of primary renal tuberculosis, the cystoscope shows the corresponding ureteral opening ulcerated. If the correct diagnosis be established early, and nephrectomy carried out promptly, these ulcerations around the vesical end of the ureter disappear. It is to be expected that the same will happen with tuberculous infiltrations or ulcerations farther up within the canal; in other words, that the process within the ureter heals.

In a very interesting case of this class I saw the formerly much-diseased ureteral mouth fully restored four months after nephrectomy. The patient, a woman of 46 years, had been sick with urinary symptoms not quite a year. She is absolutely cured and well up to date.[1]

It seems to me that the cystoscope might often furnish the best means for determining the indication for secondary ureterectomy in tuberculous cases. If these ulcerations of the ureteral mouth and in its immediate neighborhood persist after *an early nephrectomy* on that side, we may conclude that the tuberculous process within the ureter has no tendency to heal. On pressing upon the hypochondriac region during cystoscopy, we shall then probably also observe the entrance of pus from the ureter into the bladder. I think that we can rely on these cystoscopic facts.

Another indication for secondary ureterectomy in tuberculous cases may arise, if the original wound, which had thoroughly healed at first, breaks open again and a sinus establishes itself.

A few months ago, a lady was sent to me by one of the leading surgeons of this city with the request that I establish the presence or absence of a tuberculous process in a ureter, which had been left behind after nephrectomy. The operation had been done by a surgeon

[1] *Medical News*, May 1, 1897.

outside of New York, for — it was stated — tuberculosis of the left kidney associated with stone. Lately the lumbar wound, which had at first closed firmly, had reopened, and the lumbar fistula annoyed the patient. Her physician at home had diagnosed: Continuation of the tuberculous disease within the ureter of the formerly affected side. He advised immediate removal of the whole canal, an operation which was much dreaded by the patient, and for which she was, for special reasons, ill prepared at that particular time. Her doctor had insisted on the advisability of immediate interference, however, and sent her to New York for that purpose. The questions which I was asked to solve were: Is the left ureter diseased? If so, is it tuberculous? Does the pus in the urine come from that ureter or from the right kidney? The cystoscope showed a rather healthy bladder, and the left ureteral mouth not ulcerated; but a flake of pus rested in its lumen and projected into the fluid within the bladder, swaying to and fro. On pressing over the course of the left ureter from without, a few more drops of thick pus were seen to slowly enter the bladder and drop into the fundus. In view of the suppuration within the ureter, but the total absence of ulcerations around the ureteral mouth and in the bladder, in view also of the extreme rarity of the combination of the two pathologic processes in the kidney — tuberculosis and stone,[1] — I expressed the opinion that the removed kidney had probably not been tuberculous, but a pyonephrotic stone kidney, and that now, perhaps, an impacted stone or a stricture of the ureter was the cause of the intra-ureteral suppuration. I thereupon advised: if the lumbar fistula should persist in annoying the patient by its discharge, to cut down on the ureter at a time convenient to the patient and remove the stone; or, if the ureter should be found more seriously diseased, or if a stricture should be the cause of the trouble, to extirpate the entire canal. On microscopic examination of the pus discharged from the lumbar sinus, done later, tubercle bacilli could not be detected, nor could their presence be demonstrated by urinary analysis. The further development of the case up to date seems to prove the correctness of my conclusions.

In none of my cases of renal tuberculosis, where the patients died months or years after nephrectomy, could the cause of death be attributed to the fact that a probably diseased ureter had been left behind. Nevertheless, the statement that the ureter of a tuberculous kidney, provided the trouble had been diagnosticated and attacked *early*, can generally be safely left behind, is not conclusive by any means. Only

[1] I have observed but one case in which I found an exceedingly small concretion within a tuberculous renal abscess.

carefully conducted autopsies of patients who died after nephrectomy for tuberculosis, without ureterectomy having been added, will clear up this question. To-day we can say this much, however, that total extirpation of the ureter in combination with removal of a tuberculous kidney is indicated only if the tube is visibly enlarged and suppurating.

3. *Pyonephrosis and Pyonephrotic Stone Kidney.* It was the operation performed on a case belonging to this class that induced me to write this article. I will discuss this indication for total extirpation of the ureter in the course of my statement of the history of the case.

G. H., 23 years of age; born in the United States, has always been perfectly well in regard to his urinary system, until five years ago when, suddenly, without any apparent reason, he was seized with lumbar pain, more pronounced on the left than on the right side. A few days later a rather severe hæmaturia set in. Blood was found to be intimately mixed with the urine. He passed blood for six to seven weeks. After that a good deal of mucopurulent deposit was found in the urine. The hæmaturia never returned, but the turbidity of the urine continued. About two years ago micturition became more frequent; the patient urinated every two hours during the day, not at all during the night. The act of micturition was not accompanied with pain. There was no tenesmus. He had never passed a stone.

When first seen by me in June, 1897, he urinated four to five times during the day, with some pain at the neck of the bladder during the act of micturition; nights, but once. Of late he had noticed more sediment in his water, and occasionally pains in the left lumbar region, which travelled down towards the bladder. Before that the urine had been quite clear at times, and he had had less trouble and pain. The patient's father had died from phthisis, also his mother, and an uncle and an aunt from his father's side. The patient himself had often had pneumonic attacks during childhood. He never had gonorrhœa. During the last two years he had been at times seized with what appeared to be epileptiform attacks. The patient is a well built, rather muscular young man. On palpation both kidneys appear free from pain on pressure; prostate is sensitive, its right lateral lobe somewhat larger than the left. On massaging the same per rectum, some viscid fluid is discharged through the meatus. The patient maintains that a similar discharge has been frequently observed lately after defecation. *Urinary analysis* shows: Urine, acid, contains ¾ per mille albumin, few blood cells, little mucus and pus, few hyaline and epithelial-studded casts, no tubercle bacilli, cells of superficial and middle layer of bladder, also groups from renal pelvis. Diagnosis based on this analysis: Hyperæmia of the parenchyma of the kidneys with irritation and slight catarrh of the pelvis; possibly a more serious affection of one kidney with compensatory working of the other.

July 3. *Cystoscopy*: Multiple hyperæmic, slightly elevated spots, disseminated over the whole vesical mucosa. On the left lateral wall, not far from the ureteral mouth, a scarlet-red, diffusely outlined spot with some adherent mucus is detected. The right ureteral mouth shows distinct contours. In its immediate neighborhood the mucous membrane appears normal, of almost bluish-white color. Many injected vessels run from the ureteral opening into the neighboring mucosa. The right ureteral fold, as well as the interureteral bar, are very prominent, and not diseased. The left ureteral mouth appears swollen and succulent, less sharply defined than the right, ragged, especially towards the median line, not greatly injected. The surrounding mucous membrane is in the same swollen condition, not ulcerated. The left ureteral fold is not as strictly defined as on the opposite side.

In view of the history of the patient, the trouble was open to the suspicion of tuberculosis. But tubercle bacilli had not been found on urinary analysis; cystoscopy revealed the left ureteral mouth to be swollen, not ulcerated. The other alternative was stone in the left

kidney. On account of my pending summer vacation, the patient was, for the time being, put on a milk diet, increasing doses of guaiacol, and sent to the country. When seen again in the fall, his condition was unchanged. The urine appeared as turbid as before. There were often pains in the region of the left, on and off also in that of the right kidney. Frequency of micturition : six to eight times during the day, once or twice during the night. *Cystoscopy* was repeated with the intention of catheterizing the ureters. Intravesical appearance is little changed. The right ureteral mouth and its immediate neighborhood is in the same condition as it was found four months ago ; the left ureteral fold is more prominent ; it projects like a small tumor into the bladder. Its mouth appears immovable during several minutes of inspection. No whirls of emerging urine are observed as on the other side. A mucous thread projects from the lumen of the same into the bladder ; it sways slightly within the fluid that had been injected into the viscus previous to examination, but it is not pushed off, as is generally seen effected by the jets of intermittently descending urine. *Catheterization of the ureters with Casper's instrument :* It is easily accomplished on the right side where the tiny catheter is pushed up into the ureter for about one inch to an inch and a half, and 10 cc. of clear urine are collected in 20 minutes. The patient had not ingested any special amount of liquid before the examination. The left ureteral mouth is also easily entered, but the catheter cannot be introduced for any distance ; about one half inch up a distinct obstruction is encountered. On pushing the catheter from without it is seen to be bending at the ureteral opening. Separate drainage of the left kidney is impossible. As the patient had passed 250 cc. of a rather turbid urine just before the examination, this amount together with that drained from the right kidney was sent to Dr. Sondern's laboratory for examination. *Report on bladder urine :* Acid, specific gravity 1016, moderately marked deposit ; amount of urea, 0.019 gm. in 1 cc.; trace of albumin ; chlorides approximately 0.005 gm. in 1 cc.; moderate excess of phosphates ; no blood ; small amount of pus and mucus ; some hyaline casts ; no tubercle bacilli ; numerous cells of the superficial and middle layers of the bladder ; considerable amorphous urate deposit. *Report on specimen per ureter catheter from the right kidney :* Acid, specific gravity 1023 ; amount of urea, 0.025 gm. in 1 cc.; trace of albumin ; chlorides approximately, 0.01 gm. in 1 cc.; no excess of phosphates ; small amount of blood (from use of catheter) ; no pus, mucus, casts, or bacteria ; numerous cells of ureter ; considerable amount of amorphous urate deposit. *Conclusions* : Right kidney normal ; left kidney : at least a hyperæmia of the parenchyma and probably a more marked lesion, with compensating excretory action of the other kidney ; with at least a catarrh of the renal pelvis and probably a moderate pyelitis ; chronic cystitis.

On basis of these facts the patient was advised to enter the hospital for nephrotomy, probably nephrectomy on the left side. I expressed the view that the pyelonephritis was probably due to stone ; that there also was a stricture of the left ureter near its vesical end, the result of former ulceration, or due to an impacted stone. The possibility was mentioned further, that in spite of the repeated absence of tubercle bacilli in the urinary specimens, the cause of the trouble might be tuberculous.

November 20th. Nephrotomy at the German Hospital. Simon's incision. Kidney large, hyperæmic, with fluctuating pelvis, and dilated upper portion of the ureter. No tubercles are found on the kidney surface. The organ is easily brought in front of the lumbar incision. No stone is palpable. Aspiration of the pelvis demonstrates the presence of a slightly turbid fluid. On incising the pelvis, about three ounces of the described fluid run out. The finger introduced fails to detect a concretion. The calices are enlarged. To make things absolutely sure, the renal vessels are compressed by an assistant's fingers and the kidney is incised at its convexity and split in two halves, down into its pelvis (section cut). There is no stone in the parenchyma either. The renal tissue appears not materially altered. The two halves are stitched together with four to five catgut sutures ; the digital compression of the pedicle is then interrupted. The consequent slight oozing at the site of the incision is easily controlled by pressing some aseptic gauze on the wound for a short while.

The cause of the hyo-pyonephrosis, then, was not found in the kidney itself. It had to be looked for farther down. The kidney was pushed back into its normal bed, the edges of the pyelotomy-wound were held apart with sharp retractors and the ureter explored. A flexible rubber bougie met an insurmountable resistance about six to eight inches below the pelvis. On introducing a small soft-rubber catheter, cut off transversely at its end, and throwing in sterile water with a hand-syringe, it was seen that about one to two ounces would flow down without re-appearing. But on increasing the quantity of the injected fluid, suddenly the whole amount was regurgitated. The procedure was repeated, to make sure of the observed phenomenon. The catheter was withdrawn from the ureter, and a long metal probe was introduced instead. It met the same firm, but soft obstruction. There was no feeling of grating. *Conclusions :* Stricture of the ureter with accompanying hyo-pyonephrosis. The causative factor was so far unknown. Perhaps, in view of the result of the ureteral catheterism, a stone which had travelled down, and was now impacted in the ureter near the ureteral mouth, had formerly set up an ulceration with consecutive formation of a stricture at that spot. In consideration of the comparatively good appearance of the renal parenchyma, at least macroscopically, I could not at the moment make up my mind to remove the kidney. Accordingly the pelvis was drained, the remaining wound tamponed with aseptic gauze, and the patient brought to bed. On the following day, I explained the condition to the young man. I stated that I could try to resect the ureteral stricture and perform a uretero-ureterostomy, and thus succeed probably in saving the kidney ; but that I could not promise a cure. On the other hand, the treatment might be considerably shortened by removing the kidney, an operation which could still be easily accomplished, and was justifiable, as we knew from the previous separate drainage that its fellow was present and healthy.

The patient decided to have the left kidney extirpated. On November 24th, the gauze was removed from the wound, a strong clamp applied to the renal vessels with ureter and the kidney cut off. The wound was sutured at its ends, otherwise left open and drained with strips of sterile gauze. The clamp was removed after forty-eight hours. Recovery was uninterrupted. Early in January, 1898, the patient was discharged with his wound healed, and much improved in every respect. I then lost sight of him.

Fifteen months later, in March, 1899, my friend, Dr. Herm. Goldenberg, called on me to inquire what kind of an operation I had performed on G. H. The young man had come to his department in the dispensary of the Mt. Sinai Hospital, complaining of pain in both lumbar regions, frequent turbidity of urine, and general malaise. Separate collection of the urine with Harris's segregator on two occasions had shown the descent of clear urine from the right side and about one to two ounces of turbid, purulent urine from the left. This result was inexplicable, inasmuch as the patient maintained that his left kidney had been removed. In answer to the inquiry, I explained that the left kidney had been removed, but that its strictured ureter had been left behind. I called attention to the possible inaccuracy of the result obtained with Harris's instrument. The latter was at that time on trial in New York in the hands of many colleagues. The doctor promised to repeat the examination for a third time. If the same misleading observation should be made, we agreed that the bladder should first be thoroughly flushed until the water returned clear ; a specimen of this clear fluid should be put aside. Then the same amount of clear water should be thrown into the bladder, the catheter, plugged, left in place, and massage performed for a short while along the diseased left ureter. If the water in the bladder should become turbid, it would be proven that the site of the suppuration was within the left ureter. A third trial with Harris's instrument again allowed about 10 cc. of a turbid urine to run from the left of the intravesical watershed. The other experiment gave a positive result. Having later satisfied myself as to the correctness of the massage-test over the left ureter, the indication for ureterectomy was clearly established. On March 18th, it was done at the German Hospital, with the incision as devised by James Israel, of Berlin. The cut was made through the abdominal wall from the tip of the eleventh rib towards a place corresponding to the middle of Poupart's ligament.

The ureter was found to be about the size of the thumb, soft, fluctuating, and covered with a dense network of small blood-vessels. After careful dissection an additional incision was made at the upper end of the wound, parallel to the twelfth rib, into the scar that had resulted from the former nephrectomy, and the wound at its lower end continued to the border of the left rectus muscle. By turning the patient on his right side, and placing him in a Trendelenburg posture of various degrees, I well succeeded in exposing the entire canal down to the bladder without ever entering its cavity. It was clearly visible from without that there was a tight stricture at the junction of about the middle and lower thirds of the ureter. When about to tie off the lower portion at its entrance into the vesical wall, a stone of about the size of a large pea was found firmly wedged in the ureter, just above the ureteral mouth. After some efforts it was successfully dislodged and pressed into the ureter towards the lower aspect of the stricture. Then the upper and lower ends of the ureter were tied off with strong catgut, and the whole organ removed by cutting it off between these ligatures and a clamp, which had been applied about half an inch away from each ligature. In order to catch a possibly outflowing drop of the infected contents of the ureter, aseptic gauze had been pushed below the organ, near the bladder. I thus succeeded in keeping the large retroperitoneal wound absolutely aseptic. The lumen of each stump was then cauterized with the Paquelin, a strip of sterilized gauze run down to them, and the entire abdominal wound carefully closed layer by layer with continuous catgut sutures. The patient made a perfect and uninterrupted recovery. To-day, ten months after the operation, his abdominal wall is firm everywhere; there is no tendency to hernia. Figures 1 and 2 [1] show the extent and present appearance of the scar. The specimen, when cut open, presented a very narrow stricture at the junction of the middle and lower thirds of the canal, as stated. The part above as well as below it was much dilated (see Figure 3). [2] The stone had a number of sharp projections all over its circumference (Figure 4). They had, no doubt, caused its firm lodgment within the vesical end of the ureter, just within its narrowest spot, the mouth.

FIG. 3. Extirpated ureter (½ natural size), showing tight stricture at junction of middle and lower third, with marked dilatation above and below.

FIG. 4. Calculus (⅔ natural size), that had been wedged in the extreme vesical end of the ureter.

With these pathologic specimens at hand, the history of the case is easily explained: Five years ago this stone entered the ureter, producing pain and the hæmaturia. At its entrance into the small pelvis, it was arrested, causing the ulceration, followed by stricture. Later the stone itself travelled farther down, but was again held up right in front of the ureteral mouth on account of its irregular surface. This second stricture in the course of the ureter, represented by the stone, caused the dilatation of its lower third. That it was this one stone only that gave rise to the many years of sickness is proven by the fact that the patient at no time had symptoms pointing

[1] I am indebted for these photographs to Dr. E. Wettengel, of the house staff of the German Hospital.

[2] This drawing was kindly made for me by Dr. Herman Fischer, of New York, former

Fig. 1.

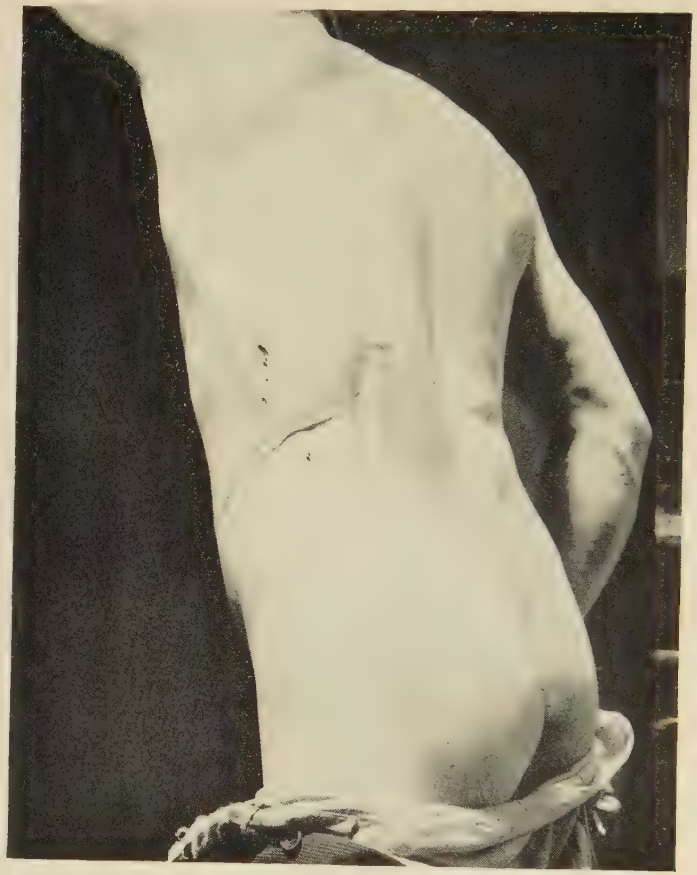

Fig. 2.

to the presence of a vesical stone, nor did he ever pass any. To-day the patient voids perfectly clear urine at normal intervals. His left side never gives him trouble; at times he experiences pain in the right kidney. Perhaps nephrolithiasis, so often found bilateral, is now developing on his right side (?). He is long back at work, but often annoyed by his epileptic seizures.

This case nicely illustrates what an amount of mischief can be produced by a single renal calculus, which cannot pass the ureter. It also shows the necessity of carefully testing the patency of the lower portion of the ureter when performing uretero-ureterostomy for stricture. Had my patient consented to this conservative operation, we probably might have saved his kidney; yet, had I then omitted to satisfy myself as to the rather improbable presence of a second obstruction farther down, even the successful restoration of the ureteral lumen would have been a failure functionally. On the other hand, it would probably not have been difficult, at the time of such an operation, to mobilize a stone, lodged within the vesical end of the ureter, and to push it up in order to be extracted through the ureteral incision, before the canal was again closed.

The case further demonstrates the necessity of always testing the patency of the ureter when performing nephrotomy and nephrectomy. By doing so a stricture will be definitely diagnosticated and located. If a grating is felt at the point of resistance, the presence of a stone lodged within the ureter is demonstrated. If the stone can be reached from the lumbar wound, the surgeon ought to try and push it up and extract it. If, however, the calculus is in the lower end of the ureter, the organ ought to be exposed extraperitoneally at once, provided the patient's condition permits of such a procedure. If it does not, it must probably be done later to avoid further trouble. In a case of nephrectomy for pyonephrotic stone-kidney I lately succeeded in nicely pushing up and extracting two irregular stones from the upper part of the ureter. Free irrigation downward into the bladder proved afterwards that the canal was unobstructed farther down. If a ureter is filled with a great number of calculi, it is best to excise the entire canal.

I do not believe that a stricture in the ureter without the presence of the stone, whose passage originally caused the stricture, will often necessitate the extirpation of the ureter after the kidney has been removed. It is rarely so tight as to prevent the small amount of mucus discharged above from passing into the bladder. If diagnosticated during nephrectomy we ought not to tie and cauterize the ureteral stump, but rather leave the ureter open and stitch it into the lumbar wound. With proper

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house surgeon of the German Hospital, who assisted at the operation. It was made very soon after the operation, from the specimen.

drainage of the upper part of the ureter into the lumbar wound and, if necessary, with irrigation and dilatation of the ureteral stricture from this wound, the ureter has a better chance to heal.

Since writing this article I have done a second total extirpation of the ureter in a man, thirty-seven years of age, who had his left kidney extirpated by another surgeon for what seems to have been a pyonephrosis, almost four and a half years ago. After this his general health improved, but not the local trouble. Four months later his bladder was drained through the perineum for a number of weeks, by the same surgeon; no improvement. One year later suprapubic cystotomy was done, this time in another hospital, without, however, improving his condition. A fistula remained at the site of the incision. Lately he has come under my care. On examination I felt a hard cord in the course of the left ureter, very painful on pressure. A valve-like obstruction at the neck of the bladder prevented the entrance of an instrument, even of small size. Vesical irrigation and cystoscopy were, therefore, impossible. With the view of making as distinct a diagnosis as was possible under the circumstances, I made him pass some urine. The same was turbid but odorless. Then I massaged the left groin downward. The urine then voided was entirely different in color, contained much more pus, and was very offensive. The diagnosis of chronic suppurative ureteritis was established beyond a doubt. On January 4, 1900, I extirpated the diseased ureter. The operation proved to be extremely difficult on account of the old and very firm adhesions around the very much enlarged and soft-walled canal, which had been cut off pretty far below the renal pelvis at the time of the first nephrectomy. I succeeded, however, in removing the entire ureter down to its entrance into the bladder. At this latter spot it was strictured, but did not contain a stone. The patient has made a good recovery and will soon leave the hospital.

By making it a rule always to test the patency of the ureter down into the bladder when doing a nephrotomy or a nephrectomy, and then, according to the result of this examination, adding, immediately or later, the necessary treatment of or operation on the ureter, many a kidney will be saved from extirpation and much trouble be spared to our patients.

CARDIAC MALFORMATION WITH AN UNUSUAL ARTERIAL DISTRIBUTION, ACCOMPANIED BY A SYSTOLIC MURMUR WHICH WAS LOUDEST POSTERIORLY.

By L. EMMETT HOLT, M.D., New York.

J. O., nineteen months old, was admitted to the Babies' Hospital, October 10, 1899.

Patient was the youngest of six children, four of whom had died from diphtheria. The father was reported to be suffering from tuberculosis.

The mother stated that nothing unusual had been noticed in the child at birth, but during early infancy he had had a blue attack, during which a physician examined him, and said he had heart disease. The child is said to have had a number of such attacks, but to have suffered from no constant cyanosis. He has never been well cared for and has always been a very delicate child.

On admission to the Hospital he was found to be much below the average in development. His weight was 17 pounds; his length 29½ inches; circumference of the head 18 inches; circumference of the chest 17¼ inches; 16 teeth, partly decayed. He was moderately rachitic, there being a slight chest deformity and well-marked rosary; the fontanel admitted the tips of two fingers; spleen and liver both enlarged, and the lower border of each extending one inch below the border of the ribs. There was no clubbing of the fingers, no cyanosis, and no œdema.

Heart.—The apex beat was in the mammary line one inch below the nipple; in front, there was a moderately loud systolic murmur, audible over nearly all the left chest with maximum intensity near the apex of the heart. Behind, a systolic murmur was heard upon both sides of the chest, the loudest point being on the left side near the spine, where it was considerably louder than in front.

The patient was under observation until his death, six weeks later, giving an opportunity for repeated examinations of the heart during

this period. At no time did the signs differ materially from those present on admission. These were:

1. In front, a systolic murmur heard over the whole left chest, the point of maximum intensity being, as a rule, about midway between the nipple and the median line.

2. At times, but not constantly, there was heard a double murmur near the apex of the heart, and in the left axillary region.

3. The aortic second sound was intensified.

4. A systolic murmur was heard behind over both sides of the chest, but was much louder on the left side than on the right, the point of greatest intensity always being near the spine at the level of the sixth to the eighth dorsal vertebra.

5. The position of the apex beat was in the mammary line one inch below the nipple.

6. There was marked epigastric pulsation.

The cardiac dulness did not extend beyond the right border of the sternum; there was no marked venous pulsation in the neck. The systolic murmur heard in front became somewhat louder at times than on admission, but it was never so loud as the murmur heard behind.

About one week after admission, the patient developed signs of broncho-pneumonia in both lungs, and consolidation appeared in the right upper lobe, which continued until death. Acute symptoms with fever lasted for two weeks, during which time, and for a period afterward, general cyanosis was present, but was never intense, the skin being of a dull gray, but never of a deep purplish color. At the same time there was marked œdema of the face, and slight œdema of the feet, without any abnormal condition of the urine. It was apparently of cardiac origin. Both the œdema and the cyanosis disappeared entirely about two weeks before death. This occurred from progressive asthenia.

AUTOPSY.

Heart.— The organ was slightly larger than normal and somewhat square in its general outline (Figure 1). The aorta was very much enlarged and measured one inch in diameter. The pulmonary artery was small and placed somewhat behind the aorta, being only one quarter inch in diameter at its origin, but widening to one half inch at a short distance from this point.

The right ventricle was greatly hypertrophied and occupied almost the entire anterior surface of the heart. Its walls were slightly thicker than those of the left ventricle.

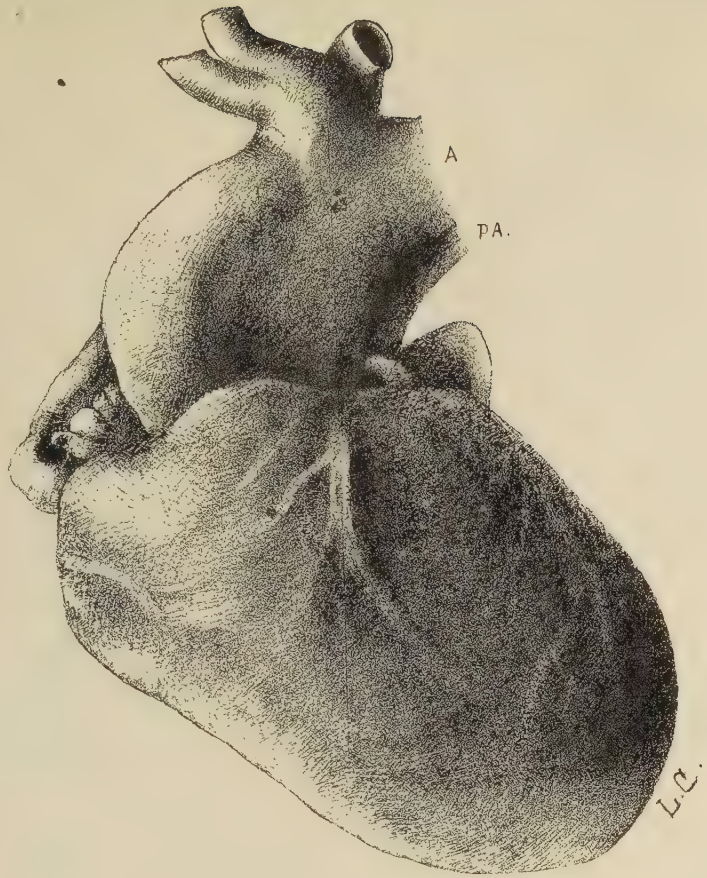

FIG 1.

Malformation of the Heart; anterior view.

A., Aorta, much enlarged; P. A., Pulmonary artery.

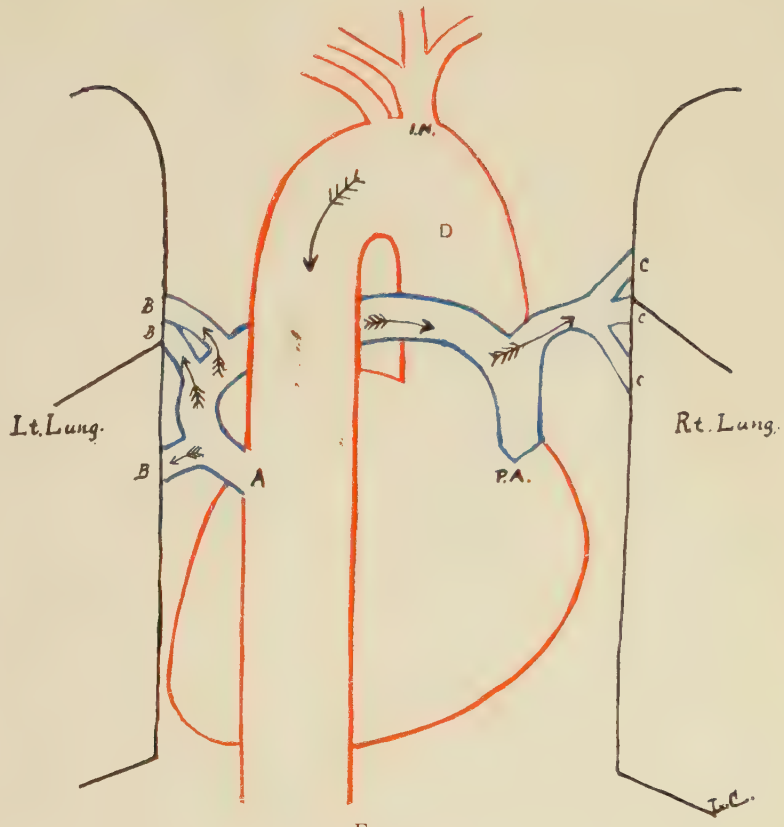

FIG. 2.

Diagram Showing Arterial Distribution; posterior view.

D, Aorta; P. A., Origin of pulmonary artery; A, Connecting branch from aorta to pulmonary artery; B B B, Branches to left lung; C C C, Branches to right lung.

The ventricular septum was deficient at its upper portion, the opening being about three quarters of an inch in diameter.

The auricular septum appeared normal.

The aorta arose directly over the ventricular septum, communicating thus with both ventricles.

The aortic valve and orifices were normal.

There was complete atresia of the pulmonary orifice. Its valves as seen from above were represented by a small nipple-like prominence composed of three distinct parts.

The mitral and tricuspid valves and orifices appeared normal.

The branches from the arch of the aorta were irregular, the left common carotid being given off from the innominate. There was no trace of the ductus arteriosus.

At the lower part of the descending portion of the arch, a large branch (A) fully one half inch in diameter was given off, through which chiefly or solely the blood reached the lungs. This branch arched across in front of the aorta, communicating with the trunk of the pulmonary artery, giving off three branches to the left and three to the right lung in the manner indicated in the accompanying diagram, which shows also the direction of the blood current.

The pulmonary veins as well as the upper and the lower vena cava were normal.

Lungs.— There was no fluid in the pleural cavity. A consolidation was present in the right upper lobe showing old caseous nodules in its centre, one of which was softened. About these nodules were evidences of old broncho-pneumonia. The right lower lobe was intensely congested and of a deep, brownish-red color. The left lung showed some spots of old broncho-pneumonia.

Liver.— Slightly enlarged, intensely congested, and showing many recent tubercles.

Spleen.— Also slightly enlarged, hard, and showing recent tubercles.

The mucous membrane of the stomach was much congested and showed a few minute follicular ulcers.

The intestines showed two or three tubercular ulcers in the ileum and one in the jejunum.

The bronchial and mesenteric lymph nodes were enlarged and cheesy.

It is not always easy, even where the clinical observation has extended over a considerable period and where opportunity is afforded after death for careful dissection, to connect satisfactorily in cardiac malformations the physical signs with the anatomical conditions. In this case there can be little doubt that the loud systolic murmur heard

behind is to be ascribed to the disturbance of the blood currents, due to the irregular vascular distribution by which the blood reached the pulmonary artery from the aorta. The murmur heard in front may have been due to the same cause, but was more probably the result of the open ventricular septum.

The entire absence of cyanosis throughout the greater part of the child's life seems rather surprising in connection with the pulmonic atresia, as it is with atresia or marked stenosis that cyanosis is most constantly associated. Its absence in this case would seem to be accounted for by the large size of the communicating vessel between the aorta and pulmonary artery; for with the hypertrophied right ventricle the pulmonic circulation seems to have been carried on pretty well, cyanosis appearing only under the stress of acute disease of the lungs.

There are few conditions in which a more complete commingling of arterial and venous blood is likely than in the case before us, so that this one affords additional evidence, if any were needed, to overthrow the theory once held that cyanosis depends upon a mingling of arterial and venous blood.

The most striking sign during life was a systolic murmur whose greatest intensity was in the circumscribed area near the spine on the left side. This, in my experience, is unique. In a collection made in 1895 of 242 autopsies in cases of congenital cardiac malformations, I found forty per cent. in which clinical histories were given. The presence of murmurs, usually systolic, was noted in four fifths of these histories, but in no case was either the lesion or the sign here mentioned, recorded. It seems hardly probable, however, that the anatomical condition is as rare as this would indicate. It is much to be regretted that in the majority of the reported cases no careful study has been made of the vascular relations, the heart having been separated from the great vessels before removal from the body, and after this has been done a satisfactory study of the vascular relations is im possible. In point of fact, this has been done in but comparatively few of the cases. If the rule were followed of removing lungs, heart, and the first five or six inches of the aorta *en masse* a study could be made of the relations of the vessels. The vascular anomalies are quite as important as and more interesting than the changes in the walls of the heart and are as yet but very imperfectly understood.

SIMPLE OR ROUND ULCER OF THE DUODENUM.

By FRANCIS P. KINNICUTT, M.D.,

Professor of Clinical Medicine, College of Physicians and Surgeons, Medical Department Columbia University.

IT is intended in the present paper chiefly to record the cases of simple or round ulcer of the duodenum which have come under the observation of my colleagues and myself, in the past several years in the wards of the Presbyterian Hospital, and to report two additional cases, one occurring in the wards of St. Luke's Hospital, and one in private practice. Since the beginning of 1893, five cases of duodenal ulcer have been under observation in the Presbyterian Hospital, in four of which, autopsies were obtained. In the fifth case the lesion was demonstrated in the attempt to save life by surgical interference. In the remaining two cases autopsies were also obtained. During the period dating from 1893, a diagnosis of ulcer of the duodenum was made in five other cases under observation in the Presbyterian Hospital, and in private practice (one case). Four of the cases recovered, and in the fifth, terminating fatally by hemorrhage, an autopsy was not permitted. In this case, the clinical history strongly suggested a tuberculous origin of the lesion.

Although the cases in which simple or round ulcer of the duodenum was found to be present at autopsy add little to our knowledge of the etiology, the symptomatology as distinguished from that of peptic ulcer of the stomach, or the pathogenesis of the disease, their histories, nevertheless, contain many points of interest.

The frequency of simple ulcer of the duodenum has been variously estimated by different authors. It is evidently impossible to form an accurate estimate from the number of cases diagnosed as such during life, on account of the great uncertainty of the diagnosis. The largest number of post-mortem records which have been studied for the purpose of eliciting this point are those of Perry and Shaw,[1] published in *Guy's Hospital Reports* for 1893. In 17,652 autopsies made at Guy's Hospital from 1826 to 1893, seventy

cases of duodenal ulcer, open or healed, were found—that is, in .4% of persons dying from all causes. In 8192 autopsies made at St. George's Hospital, London, between 1863 and 1893, Latham[2] found twelve cases of perforating ulcer of the duodenum, therefore in .14% of persons dying from all causes. The number of cases of non-perforating and healed ulcers apparently was not investigated. Turning to Continental statistics, in 5000 autopsies at the Pathologico-Anatomical Institute of Prague,[3] six cases of duodenal ulcer, .1%, are recorded. Plange,[4] Steiner and Wollmann[5] collectively, report twenty-two cases in 4802 autopsies, .45%. In 1150 autopsies made by Grunfeld,[6] in Copenhagen, four instances of ulcer of the duodenum, .34%, were observed. In 1124 autopsies made at the Presbyterian Hospital, New York, from January 1, 1893, to December 31, 1899, perforating and non-perforating ulcers were present in four cases, in .35% of persons dying from all causes. The fatal case in which this lesion was discovered at operation, an autopsy not being permitted, would increase the percentage to .44. Undoubtedly, cicatrices of healed ulcers in the duodenum are frequently overlooked, and in several of the statistical records quoted, it is not especially stated that the number of cases includes cicatrized ulcers. In the large number of post-mortem records studied by Perry and Shaw,[1] the seventy cases include all duodenal ulcers, open or healed. The percentage agrees so closely with that of the Berlin, Copenhagen, and our own records, that it would seem justifiable to conclude that simple ulcer of the duodenum, cicatrized or open, is found in about .4% of persons dying from all causes.

The pathogenesis and pathology of duodenal ulcer correspond in their main features with those of gastric ulcer. The digestive action of the gastric juice is universally accepted at the present time as an essential factor in its pathogenesis. The situation of the duodenal lesion, almost without exception between the pylorus and the biliary papilla, above the point of neutralization of the gastric juice by the pancreatic secretion, hardly admits of any other explanation. The frequent coexistence of gastric and duodenal ulcers, presenting similar anatomical characteristics, further corroborates this view. The hypothesis of a localized antecedent impairment of nutrition, variously produced, of the mucous membrane, as a necessary pathogenetic factor, is equally accepted. A hyperacidity of the gastric juice, which obtains very generally in gastric ulcer, has not been demonstrated in a sufficient number of cases to admit of a judgment in regard to its etiological importance. Diminished acidity has been observed in a certain number of cases. The features in which duodenal ulcer especially differs from ulcer of the stomach are of much interest, and their explanation is

still largely hypothetical. They are the comparatively frequent occurrence of duodenal ulcer in the new-born; the greater frequency of the lesion in males than in females, the ratio being estimated by Collin[7] as 3.9 to 1, a ratio the reverse of that which obtains in gastric ulcer, which is three females to two males; especial pathological conditions, with which duodenal ulcer has been observed to be associated, and which are variously considered as predisposing causes; and, finally, the frequency of certain complications of the gravest character.

The duodenal ulcer of the new-born presents the anatomical characteristics of the lesion in the adult. Severe hemorrhage is a common accompaniment, giving the earlier name, melæna neonatorum, to the disease. From its occurrence in new-born children who have lived only a few hours, an intrauterine origin has been surmised. Landau's hypothesis,[8] based upon the observation by him of two cases with autopsies, that the lesion is due to thrombosis of the umbilical vein with embolism and consecutive necrosis of a circumscribed portion of the mucous membrane, is probably correct. Boas has suggested an ingenious hypothetical[9] explanation of the greater frequency of ulcer of the duodenum in males. He ascribes it pre-eminently to the use of alcohol and tobacco, basing his hypothesis upon the well-known experimental investigations of v. Mering and Moritz on dogs. It has been shown by them that the stomach expels water into the intestine with great rapidity; and further that alcohol, salines, dextrine, and acids, although absorbed by the stomach, are taken up by it to a very limited extent; that the water first discharged contains these substances in a very concentrated solution; and that the fluid portions of the chyme are first released by the stomach, the solid portions following more slowly. The duodenal membrane is thus much less protected against concentrated solutions than the stomach, whose mucous membrane is relatively protected during the first stage of digestion by the porridge-like or solid chyme.

It is therefore through the action of concentrated acid, saline, and alcoholic solutions upon the duodenal membrane, already irritated by a presumably hyperacid gastric juice, that a necrosis occurs. It is not clear whether Boas believes that the concentrated solutions *per se* are capable of producing that impairment of nutrition which has been accepted as a necessary antecedent to a peptic digestion of the mucous membrane. The views of clinicians vary in regard to the etiological importance of alcohol. My own observations would lead me to regard its excessive use as a distinct predisposing cause of duodenal ulcer. It is quite certain that its abuse may precipitate the gravest symptoms.

In two of the cases whose histories are appended, there had been an excessive alcoholic habit for years, and in three of the remaining seven, a moderate habit. In Case 2, acute symptoms had several times occurred on especial excess, and in both Case 2 and Case 5, a sudden development of the complications which resulted fatally occurred during a period of excessive drinking. Among the associated pathological conditions found in duodenal ulcer, burns of the external surface and septicæmia are of especial interest. A causal relation would seem to be borne by them. Especially true is this of burns, and various theories have been advanced in explanation of the relationship.

Circulatory disturbances which are conspicuous at least in the form of congestion and ecchymoses throughout the course of the intestinal tract in burns of the external surface, in connection with the conditions favorable for auto-digestion which exist in the upper portion of the duodenum, form the basis of these hypotheses.

The formation of thrombi and emboli through the liberation of a soluble fibrin ferment, hyperæmia and ecchymoses due to a diminution of vascular tone, a reflex effect of excessive irritation of the nervous system, congestion and ecchymoses due to the excretion through the bile of toxins of an irritating character absorbed from the injured skin (Hunter),[10] are variously believed to represent the nature and the cause of the circulatory disturbance. Billroth[11] believes the lesion to have its origin in a special form of septicæmia, and Perry and Shaw[1] regard a septic condition of the burnt surface as the direct cause of the ulcer. It is interesting to note in this connection that the Guy's Hospital records indicate that duodenal ulcer is associated with a general septic or pyæmic condition nearly as frequently as with burns. It is also a fact that cases of duodenal ulcer following burns are much rarer in the literature of the past ten years than earlier, that is, with improved methods of treating wounds. The 17,652 autopsies at Guy's Hospital give a ratio of 18 duodenal ulcers, associated with septic conditions other than burns, to 52 ulcers from all other causes. In 10 of 21 cases collected by Perry and Shaw,[1] there were sloughing conditions of the skin or cellular tissue. The most reasonable explanation of the relation between the duodenal lesion and septic infection would seem to be in the occurrence of small extravasations of blood in the duodenal mucous membranes such as are commonly observed in the serous and mucous membranes in general sepsis, due as a rule to infective emboli. The subsequent digestive action of the gastric juice would complete the process. The rarity of gastric ulcer both in burns and in general septic or pyæmic conditions remains unexplained. Among other diseases with which duodenal ulcer has been found associated, and variously

regarded as predisposing causes, are nephritis, tuberculosis, cardiac disease, erysipelas, trichinosis, pemphigus, and frost-bite. That the association of nephritis may be regarded as more than a coincidence is suggested by the statistics of Perry and Shaw.[1] In the 70 cases of duodenal ulcer occurring in Guy's Hospital, nephritis was present in 12 cases, 16%, whereas some form of nephritis existed in only 7% of persons dying from all causes in the Hospital.

Boas[9] believes that the influence of the renal disease in producing the lesion resides in the necrotizing effect of the urea or its derivative ammonium carbonate retained in the blood current. A more reasonable explanation in many cases would seem to be in an endarteritis or sclerosis of the duodenal vessels.

In the cases of duodenal ulcer associated with advanced pulmonary tuberculosis which I have been able to collect, the lesion is described in many instances as presenting all the anatomical characteristics of peptic ulcer. In several, perforation occurred. In some cases the ulceration was confined to the duodenum, in others a typical peptic ulcer was present in the duodenum, and multiple ulcer of the ordinary tuberculous type was found in the jejunum and ileum. Although the tuberculous origin of the duodenal ulcer in such instances admits of a legitimate doubt, there are a few observations which indicate that the lesion originates in a tuberculous cheesy degeneration of a solitary follicle, with softening, and gradually acquires the characteristics of the simple ulcer through the peptic action of the gastric juice.

Although diseases of the heart, both myocarditis and endocarditis, are found in a considerable number of cases of duodenal as well as gastric ulcer, the association is not as frequent as would be anticipated in view of the theories of the important part which circulatory disturbances play in the pathogenesis of the affection. In 262 cases collected by Collin,[7] the heart was normal in 90%. This is also true of diseases in which the portal circulation is affected primarily or secondarily. In Collin's[7] cases, atrophic cirrhosis of the liver was noted only seven times. In our own cases, endocarditis coexisted with the duodenal lesion in three of the six cases in which a full post-mortem examination was made.

Single cases of trichinosis associated with duodenal ulcer have been reported by Ebstein [12] and by Klob.[13] Ebstein regards the relation as a causal one. Coexistent pemphigus has been reported by Malherbe [14] and Lignerolles [15] in two cases. Two cases, [16, 17] of duodenal ulcer and one [18] of gastric ulcer associated with frost-bite have been published. Billroth believes the relationship to be a causal one and of a septicæmic nature.

The complications peculiar to duodenal ulcer are those due to involvement of the biliary papilla in the cicatricial tissue of healing or healed ulcers. In Collin's[7] 262 observations, there were three instances of occlusion of the common bile duct from this cause. Two cases of general dilatation of the biliary and hepatic ducts almost to the surface of the liver, and two cases of stenosis of both the biliary and pancreatic ducts, were also observed. Among the serious complications common to both gastric and duodenal ulcer, due also to cicatrizing or cicatrized ulcer, dilatation of the stomach was found by Collin[7] eighteen times in 262 cases.

The complication, at once the gravest and the frequency of which most difficult to estimate with accuracy, is perforation into the general peritoneal cavity. Percentages estimated from published cases are manifestly very misleading, as perforation has been the feature which has led to the report of many cases. In Collin's[7] 262 cases, published in 1894, his statistics including all cases of simple duodenal ulcer reported since 1823, perforation occurred in 181 (69%). In the cases collected by me, comprising all cases recorded from 1894 to the present date, fifty-four in number, perforation occurred in forty-four.

Many of these cases have been reported by surgeons on account of the existence of perforation and with the view of eliciting discussion as to the feasibility of operation. The statistics of unselected cases must therefore serve as the basis for an estimate of the frequency of perforation. Among the large number of full post-mortem records previously referred to, the only ones available for such a purpose are those of Perry and Shaw[1] and of the Presbyterian Hospital. In the 1124 autopsies at the Presbyterian Hospital from 1893–1900, duodenal ulcer was present in four cases (.35%), and perforation existed in two of these. Including the fatal case in which the lesion was discovered at operation, perforation occurred in 60%. On the other hand, in Perry and Shaw's[1] seventy unselected cases of duodenal ulcer, perforation was present in only eight (11%).

The comparatively small number of autopsies in the Presbyterian Hospital records may be regarded as militating against their acceptance as a correct basis for estimation, although the ratio of cases of duodenal ulcer to the total number of autopsies almost exactly coincides with that of the Guy's Hospital statistics. If Perry and Shaw's[1] percentage is even approximately correct, the frequency of perforation into the peritoneal cavity is nearly twice as great as has been estimated in the case of gastric ulcer.

My inference from all available data coincides with that of Welch,[19] who estimates the frequency of perforation in gastric ulcer to be about 6.5%.

It is of much interest to note that in 104 cases of Collin, where the site of the perforating ulcer is specified, in sixty-nine instances it was on the anterior wall of the duodenum, and in thirty-five on the posterior wall.

Varied opinions are held as to the possibility of the clinical differentiation of duodenal from gastric ulcer. Nothnagel, Leube, Ewald, and Collin believe that it is impossible in the majority of cases, in opposition to the views of Chvostek, Bucquoy, and Boas, among others. My personal experience is in accord with that of the former observers. The general opinion that severe gastric or intestinal symptoms are rarely observed in duodenal ulcer is based upon the apparent absence of symptoms in many instances until the perforation suddenly occurs. My experience leads me to believe that such a latency is more apparent than real and that a careful study of these cases would reveal the antecedent existence of well-marked symptoms.

Among the subjective symptoms, the character of the pain in no wise differs from that of gastric ulcer.

It is described by patients as burning or boring. *The relation* of the epigastric pain, if present, to the ingestion of food is believed by some observers to be a symptom of differential diagnostic value. They claim that in duodenal ulcer cessation of the pain, which is often present two or three hours after a meal, occurs on further ingestion of food. The explanation of this feature is found by Chvostek [20] and Burwinkel [21] in the closure of the pylorus through the irritation of food, thus preventing further irritating material from passing into the duodenum. In the cases which have come under my observation where a duodenal ulcer was found at autopsy, the more or less constant character of the pain, even when the stomach has been empty, has been a more conspicuous symptom, in marked contrast to the clinical history of gastric ulcer.

Among the objective signs, little or no assistance is given by the examination of the gastric contents. Both hypochlorhydria and increased acidity have been observed. A painful pressure-point situated to the right of the median line, between the umbilicus and the costal arch, when present, is a sign of some value. Boas has observed in several cases a painful dorsal point to the right of the twelfth dorsal vertebra.

In uncomplicated duodenal ulcer, *a priori*, vomiting would not be expected. My experience has been that it is a frequent symptom. When not due to the reflex irritation of pain, the not infrequent accompanying gastritis and the occasional gastric ectasia from cicatrizing ulcers are probable factors in its causation.

A careful study of the clinical histories of a large number of cases leads me to believe that the character of the vomited matter is of slight diagnostic value. Ewald [22] relates a personal case in which the vomited matter consisted of three totally different parts: first the food in the stomach was vomited, then bile, and finally blood. Such characteristics can be presented only occasionally.

The form of the hemorrhage, if present, is of slight differential value in my experience. In small hemorrhages in duodenal ulcer, undoubtedly the blood passes by the bowel and escapes notice; in copious hemorrhage, the clinical histories of the cases which are appended indicate that it may appear indifferently in the form of hematemesis or melæna, or both.

The prognosis in simple ulcer of the duodenum is variously regarded by different observers. Bucquoy [23] believes that it is of great gravity; Oppenheimer,[24] that cicatrization seldom occurs, is mostly incomplete, and that the prognosis is always grave. Ewald [25] considers the prognosis unfavorable. He believes that the tendency is towards deep ulceration and its attendant consequences; if healing occurs, that the cicatrices are rarely smooth and tend to contraction and stenosis. Boas,[9] on the contrary, claims that the prognosis is more favorable than in gastric ulcer, if the danger of perforation is excepted. The statistics previously quoted indicate that on the most conservative estimate the frequency of perforation is nearly twice as great as in gastric ulcer.

No more than an approximate estimate of the frequency of cicatrization in duodenal ulcer is possible. In the seventy cases of Perry and Shaw,[1] cicatrization was present in eight instances, 11 %. Kraus [26] observed cicatrization only twice in eighty cases. In Collin's [7] 262 observations, cicatrized ulcers were found twenty-three times, 8.7 %, and ulcers beginning to heal in five instances, 1.9 %. It is probable that the highest percentage given is considerably below a correct estimate, as cicatrices of the duodenal mucous membrane undoubtedly frequently escape observation.

The chief features of the seven cases whose clinical histories are related may be briefly analyzed.

Six of the cases were males, one a female. The ages were: 38 (female), 40, 34, 42, 25, 34, and 34, respectively.

In Cases 2 and 5, there was a history of an excessive alcoholic habit, and the development of the fatal attack occurred during a period of excessive drinking. In Cases 3, 4, and 6, a moderate alcoholic habit was present.

In six of the seven cases, digestive disturbances had existed for

very considerable periods, five months to many years. In Case 3, there had been a severe attack, similar in many respects to the fatal illness, six years previously.

Pain:

In Case 1, acute pain was felt only four days before death. It was located to the right of the umbilicus. Undoubtedly it immediately preceded perforation.

In Case 2, severe epigastric pain first occurred eleven days before death. It was constant. It was not localized, beyond being confined to the epigastrium. There was sensitiveness on palpation, especially marked in the right half of the epigastrium. In Case 3, little if any abdominal pain accompanied the digestive disturbance present before admission to hospital. During the five months of residence in hospital, there was almost constant epigastric pain, and of a severe character. It was present irrespective of the ingestion of food, even during the periods of rectal alimentation. There was slight diffused epigastric sensitiveness.

In Case 4, epigastric pain had been present throughout the course of the illness of two years. Its relation to the ingestion of food was not determined. After admission to hospital and during the period of eight days before death, it was constant and severe. There was marked sensitiveness on palpation over a circumscribed area to the right of the median line opposite the ninth costal cartilage.

In Case 6, the course of the ulcer was a latent one, there being no symptoms beyond comparatively slight hæmatemesis, which occurred after admission to hospital, and which was ascribed to other causes.

In Case 7, the epigastric pain was constant, and little affected by the ingestion of food during the two months of the fatal illness. There was diffused epigastric sensitiveness.

Vomiting:

In Case 1, vomiting had been a frequent symptom with an undetermined relation to the ingestion of food during the long period of digestive disturbance. The vomiting shortly before admission to the hospital probably immediately preceded perforation. In this case it is of interest that open and cicatrized gastric ulcers in addition to the duodenal ulcer were present.

In Case 2, acute attacks of vomiting had occurred in the past, during periods of excessive drinking. They were stated to be independent of the ingestion of food. Vomiting was present throughout the period of the patient's last illness.

In Case 3, there had been more or less vomiting from the beginning

of the illness. It variously occurred in the morning before breakfast, and an hour to an hour and a half after meals. It persisted until death.

In Case 4, vomiting had been a symptom throughout the illness of two years.

In Case 5, the history of "alcoholic gastritis" would indicate the probable occurrence of vomiting.

In Case 7, vomiting occurred directly after the ingestion of food of any kind, during the last several weeks of illness.

Hemorrhage:

In six of the seven cases there was no history of hæmatemesis or melæna previous to admission to the hospital. In the remaining case (1), hæmatemesis had not occurred, but tarry stools had been noticed on several occasions, three weeks previous to admission; and grumous material was vomited and passed by the bowel twenty-four hours before death. In this case, the open gastric ulcer also found at autopsy and which had probably existed for a considerable period, had not been attended with hæmatemesis.

In Case 2, profuse hæmatemesis occurred, but no melæna.

In Case 3, hæmatemesis and melæna occurred with about equal frequency during a period of five months.

In Case 4, the first hemorrhage occurred eight days before death, in the form of melæna followed by several dejections of fluid blood on the same day. On the following day, twenty-five ounces of blood were vomited, but none was passed by the bowel. In the succeeding twenty-four hours, thirty ounces of blood were passed by the bowel, but there was no hæmatemesis. On the succeeding days until death, the hemorrhage was in the form of melæna only.

Case 5 was moribund on admission to hospital.

In Case 6, there was hæmatemesis, but no melæna.

In Case 7, neither hæmatemesis nor melæna occurred at any time. The absence of hemorrhage in this case is noteworthy, as the duodenal ulcer found at autopsy was of large size (an inch and a half in diameter) and had extended to the serous coat.

In four of the cases, death was due to perforation, in one to hæmorrhage, in one to hemorrhage and inanition, in one to inanition.

In two of the cases the heart was found to be normal at autopsy; in two in which a complete autopsy was not permitted, physical examination during life gave negative evidence of cardiac disease; and in three, endocarditis existed, with mitral-valve vegetations present in two of them.

In three of the cases, the kidneys were practically normal; in two, in which the kidneys were not examined, the urinary examination during life was negative; in one, chronic nephritis was present; and in one, slight renal changes existed.

Other pathological conditions noted were advanced fatty changes in the liver in a single case.

In one case, healed and open gastric ulcers coexisted with the duodenal ulcer. In six of the cases, a single duodenal ulcer was present, in one case, two ulcers.

In three of the cases, the site of the ulcers was on the anterior wall; in one, on the posterior wall; in three, the site was not recorded.

Case 1.— Female, æt. 38. Admitted to hospital Jan. 18, 1899. Service of Dr. Andrew H. Smith.

Clinical Diagnosis: Perforating peptic ulcer, general peritonitis.

Family History: Unimportant.

Previous History: For many years the patient has suffered from time to time from indigestion and has had attacks of vomiting that apparently have not had much relation to the taking of food. She has not suffered acute pain with these attacks and has never vomited blood. Three weeks ago she passed tarry-looking matter several times in her dejections. With these exceptions she was well until four days ago.

Present Illness: Began four days ago suddenly with acute pain situated just to the right of the umbilicus. The patient also vomited several times, but there was no blood in the vomited matter. Her temperature the following day was 102°, and the abdominal pain was slightly more diffused, but there was no vomiting. She was given Epsom salts and her bowels moved several times. The temperature subsequently fell to 99.5°. Last evening, the third day of the illness, the pain became more severe and diffused, and she began to vomit a dark grumous material. She also passed by the bowels a large amount of a similar material. The vomiting and purging continued and this morning the patient felt very weak and prostrated, but the pain was not so severe. The patient was brought to the hospital in a carriage.

Jan. 18th: Physical examination on admission: The patient is moderately nourished, slightly cyanosed, and anæmic. The tongue is moist and heavily coated with a brownish fur. There is constant vomiting of a dark grumous material without odor.

Heart: The apex is in the fourth space within the mid-clavicular line. Its action is very feeble and rapid, the sounds are indistinct. No murmurs are appreciable.

Lungs: Negative.

Liver: Area of normal dulness obliterated by abdominal tympany.

Spleen: Not made out.

Abdomen: Is distended, with rigid walls. Percussion is everywhere tympanitic except in right hypochondriac and lumbar regions, where the note is dull. Over these regions and in the hypogastric region there is marked sensitiveness on palpation. In the upper right quadrant of the abdomen there is an indefinite sensation of a mass on gentle palpation.

Pulse, 140. Temperature (rectal), 104°.

The patient rapidly failed and died five hours after admission, the temperature rising to 105° immediately preceding death.

Autopsy: Anatomical diagnosis: Perforating ulcer of duodenum, gastric ulcer, general septic peritonitis.

Heart: Valves normal, muscle normal.

Lungs: There is considerable recent fibrin over the lower portion of both pleural membranes. On section, both lungs congested and œdematous.

Peritoneal Cavity: Contains a large amount of thin, dark fluid from stomach and duodenum. Intestines adherent to each other and to the abdominal wall by fresh fibrino-purulent exudate.

Liver: Consistence, very soft. Pale in color. Surface covered with fresh fibrino-purulent exudate.

Gall-bladder full of greenish bile.

Spleen: Consistence firm, color dark, considerable fibrous thickening of capsule.

Kidneys: Left: capsule free, surface slightly roughened; on section, a little opaque, markings irregular in places and indistinct in other areas. Cortex, slightly uneven in thickness. Right: capsule adherent in places, otherwise the same as left kidney. Microscopic examination. Slight increase in connective tissue. Considerable degeneration of the tubal epithelium, considerable debris in tubes.

Pancreas: Normal.

Stomach: On the posterior wall near the lesser curvature is an area thinner and more fibrous than the other portions of the stomach walls, covered by a thin and adherent mucous membrane, in the centre of which is a cup-shped depression, apparently the site of a cicatrized ulcer. Near the greater curvature are two ulcers, close together, each about one third of an inch in diameter, the mucous membrane presenting the characteristic "punched-out" appearance.

Duodenum: Just beyond the pyloric ring, on the anterior aspect of the duodenum is a perforation two thirds of an inch in diameter. The wall of the duodenum at this point is thickened and hard to the feel, and the edges of the ulcer are smooth and rounded. The peritoneal coat appears to have recently ruptured; it is ragged, sloughing, and blackened around the edge of the opening.

Case 2.—E. M. Male, æt. forty. Admitted to hospital January 10, 1899. Service of Dr. Kinnicutt.

Clinical Diagnosis: Perforating peptic ulcer.

Family History: Negative.

Previous History: Patient has used alcohol to excess for years prior to a year ago. For the past year, until two weeks ago, he has been an "abstainer." He has had gonorrhea, but not syphilis.

There is a history of digestive disturbance extending over a period of thirteen years, characterized by occasional acute attacks of vomiting similar to the present one, and by more or less constant flatulency after meals. The last acute attack occurred last July, but was only of ten days' duration. Prior to this, he had had no attack for eighteen months. The attacks usually followed a period of excessive drinking. There was never any blood in the vomited matter or in the stools, so far as the patient knows.

Present Illness: Began a week ago. The patient had been drinking to excess for a few days previously. He has suffered from loss of appetite, nausea, and vomiting independently of the ingestion of food during this period. There has been no blood in the vomited matter. There has been very constant epigastric pain, at times very acute and radiating over he abdomen. The bowels have been constipated.

Examination on Admission: The patient is well nourished and is not anæmic. The tongue is moist, but thickly coated with a yellowish fur.

Thorax: Lungs, negative. Heart, negative.

Liver : Percusses from the sixth rib to an inch below the costal arch in the mid-clavicular line, where the edge is palpable.

Spleen : Edge not palpable.

Abdomen : Not distended. It is slightly more prominent in the right epigastric region ; over this area there is a slight rigidity of the muscles, and there is much sensitiveness on palpation over the whole of the epigastrium. On deeper palpation, a mass can be felt in the right epigastric region, which seems to be continuous with the liver. Its lower border, however, cannot be palpated, and its nature is considered doubtful. The remainder of the abdomen is normal in appearance and on palpation.

Pulse 96, temperature (rectal) 100°, urine negative.

Aside from the doubtful physical signs described, the clinical history and the physical examination suggested the existence of an acute gastritis, the result of a period of excessive drinking.

Poultices were directed to be applied over the epigastrium, and small portions of peptonized milk and barley water to be given at frequent intervals.

During the three following days the patient's condition remained much the same. There was more or less vomiting after the ingestion of food, but the vomited matter consisted only of the fluids taken. Vomiting also occurred independently of food taking.

The tenderness over the epigastrium persisted, but was confined to this region. The epigastric prominence increased, but was never very marked, and the mass felt larger. The abdomen elsewhere was normal in appearance and on palpation. The temperature curve was irregular. On the 11th, the temperature (rectal) was 102° in the morning, receding to 99.5° in the evening. On the 12th, it was 100° in the morning, 102° in the evening. On the 13th, the morning temperature was 99.2°, the evening 101.5°. The bowels were moved only by enemata.

On the evening of the 13th, the third day after admission, the patient vomited a small quantity of a suspicious-looking fluid, slightly brownish in character. Physical examination revealed a marked and comparatively rapid increase in the mass in the epigastric region.

Its lower limit was palpable nearly at the umbilicus in the median line, and at this level extended to the right to a point midway between the parasternal and mammillary lines. It was firm, but not hard to the feel. The sensitiveness was still confined to the epigastrium, and to the area of the palpable mass. The remainder of the abdomen continued to be normal in appearance, and on palpation. In view of the rapid increase in the size of the mass, its situation, and the character of the vomited matter, the existence of a perforating ulcer with escape of the gastric contents into a cavity formed by adhesions or into the lesser peritoneal cavity displacing the stomach, was considered probable, and a surgical consultation was asked.

During the night the patient was very restless, and at times delirious, and vomited at intervals of two to three hours, from two to four ounces of a fluid containing a small quantity of blood altered by the gastric secretions. Suddenly, between six and seven o'clock in the morning, forty-eight ounces of fluid blood and a considerable number of largish blood clots were vomited.

The temperature rapidly fell to 96.5°, and the pulse rose to 140.

The abdomen presented no signs other than those described.

Under continuous intestinal irrigation with a normal salt solution at a temperature of 115°, and active stimulation with whiskey, nitro-glycerine, and strychnine given hypodermatically, the patient rallied sufficiently at the end of six hours, to justify, in the opinion of my colleague, Dr. McCosh, the attempt to open the abdomen with the hope of finding and closing the probable ulcer.

The patient was rapidly anæsthetized, and the abdomen opened. The stomach and liver were partially adherent. On drawing the stomach downward, a very large amount of

fluid and clotted blood was found in what appeared to be the lesser peritoneal cavity. The stomach was rapidly opened, and was found to be full of blood; the amount was estimated at more than a litre. A circular ulcer about two inches in diameter, perforating the peritoneal coat, was discovered on the posterior wall of the duodenum just below the pyloric ring.

The patient's condition was such that it was impossible to proceed further with the operation, and death occurred a few moments later.

An autopsy was not permitted.

Case 3.—Male, æt. thirty-four. Mechanic. Canadian. Admitted to the hospital, January 12, 1899. Service of Dr. Andrew H. Smith.

Family History: Unimportant.

Previous History: Inflammatory rheumatism five years ago. Venereal disease denied. There has been a moderate alcoholic habit. There is a history of gastric symptoms, very similar to those present during the past five months, six years ago, from which he recovered.

Present Illness: Five months ago he began to be troubled with eructations of gas and sour fluid from time to time, with vomiting occasionally on rising in the morning, also often an hour to an hour and a half after the ingestion of food. There was accompanying constipation, loss of weight and strength. There was apparently little, if any, abdominal pain. These symptoms have persisted up to the present date.

On Admission: The patient is fairly well nourished; is not anæmic in appearance. Pulse 74, of good force; temperature (mouth), 100.5.

Physical Examination: Negative, with the exception of eliciting slight epigastric tenderness on palpation.

Urine: 1027, acid; no albumin, sugar, or casts.

Much gastric pain is present, irrespective of the ingestion of food. There were no other symptoms beyond the vomiting of undigested food on January 15th and January 18th, until January 19th, when there were several slight syncopal attacks, which were followed on the next day by a large tarry defecation and later by vomiting of rather a large amount of altered blood. Vomiting of a similar character and melæna occurred on the 20th, 21st, 24th, and 25th. All feeding by the mouth had been discontinued on the 20th, and rectal alimentation instituted. The epigastric pain persisted during the period of rectal feeding, and necessitated the use of hypodermic injections of morphine. It was found impossible to continue the rectal alimentation beyond the fifth day, and small feedings of peptonized milk were substituted. The symptoms were not materially affected by the change; and from this date throughout a period of four and a half months, the clinical history was that of irregular, but frequent attacks of hæmatemesis, always of a grumous character, and melæna, severe epigastric pain irrespective of the ingestion of food, vomiting, marked cachexia, a hæmoglobin percentage as low as 12 at times, progressive loss of weight and strength, and an irregular temperature curve, rarely rising above 101°. Physical examinations during this period never revealed other signs than the epigastric tenderness on palpation, present on admission, and, during the last days of life, fluid in the left pleural cavity.

Various forms of fluid diet were given, and from time to time rectal feeding alone was employed and persisted in until rectal irritability compelled its disuse. The gastric contents were not examined, as it was considered unwise to introduce the stomach tube.

Death occurred on June 17th, five months after admission to the hospital, from asthenia, without the development of other symptoms than those described.

Autopsy: Anatomical diagnosis: Ulcer of duodenum, adherent pleura (right lung), left hydrothorax.

Heart: Valves normal, myo[illegible]l in appearance and on section.

Lungs: Left lung free from adhesions; the pleural cavity contains 500 c.c. of clear serum; upper lobe, emphysematous, lower lob[illegible], œdematous; right lung, a few old adhesions.

Liver and Spleen : Normal in appearance and on section.

Kidneys : Right, weight 4½ ounces ; left, 5 ounces ; capsules free, surface smooth ; on section, markings slightly indistinct.

Histological Examination : There is a very slight degeneration of the tubal epithelium, and a little granular debris in tubes.

Stomach : Slight congestion of mucosa, otherwise normal.

Intestines : In the duodenum, immediately beyond the pylorus, is an ulcer, round, with smooth edges, three quarters of an inch in diameter, its base being formed by the head of the pancreas.

Pancreas : The head is adherent to the upper portion of the duodenum, forming the floor of the ulcer.

Case. 4.—Male, æt. forty-two. Carpenter. Germany. Admitted to hospital December 26, 1897. Service of Dr. Kinnicutt.

Clinical Diagnosis : Ulcer of duodenum.

Family History : Not obtained.

Previous History : No specific history. Alcoholic habit at one time, but not of late. Inflammatory rheumatism, twelve years ago. Pneumonia, four years ago.

Present Illness : Began about two years ago with symptoms of digestive disturbance (gastric flatulence, occasional vomiting, distress and pain in upper abdomen). The most marked symptom was the abdominal pain. His statements in regard to any apparent dependence of the epigastric pain upon the ingestion of food are very uncertain. The vomiting was principally of mucus ; there was never any blood in the vomited matter. He states that the bowels have been constipated throughout his illness, and that he has never to his knowledge passed any blood in his stools.

These symptoms have been sufficiently troublesome to prevent any continuous work during the two years previous to admission.

During the past several weeks all the symptoms have been much worse ; the vomiting has been more frequent and the epigastric pain more severe.

Last evening the pain became very acute, radiating from the epigastrium into either side. This morning there was a large dejection, consisting almost entirely of dark blood. On attempting to walk to the hospital dispensary, he fell in the street and was brought to the hospital in an ambulance.

Examination on Admission : The patient is well nourished. There is marked pallor of skin and mucous membranes. Heart apex in the fifth space, three and a half inches from the median line ; sounds distant, no murmurs appreciable. Lungs, practically negative. Liver and spleen : areas of dulness normal, edge not palpable.

Abdomen : There is marked sensitiveness on palpation over an area not larger than a silver dollar, in the epigastric region, to the right of the median line, opposite the ninth costal cartilage. Over and about this area there is a marked sense of resistance, but no distinct mass can be made out. Abdomen otherwise normal in appearance and on palpation.

Urine : No albumin, sugar, or casts.

Hæmoglobin 36%. Temperature 100.5° ; pulse 124, small, and of little force. Radials slightly thickened.

Course : There was a large dejection of dark-red blood a few hours after admission, and a second similar one four hours later. On the following morning, December 27th, signs of internal hemorrhage were suddenly developed, as extreme pallor, sweating, an almost inappreciable pulse, gasping respiration, and an epileptiform convulsion. In the subsequent few hours, twenty-five ounces of dark blood were vomited at different times, but melæna did not occur.

Dec. 28th : Thirty ounces of dark blood were passed by the bowel at different periods, but there was no hæmatemesis.

Dec. 29th : There was a single tarry dejection. No hæmatemesis. Red cells, 1,640,000 ; white, 12,000.

Dec. 30th : The patient complains of much epigastric pain, and there were two tarry dejections.

Dec. 31st : No hæmatemesis, no melæna. The patient complains of soreness over the right parotid.

Jan. 1st : Red cells, 1,280,000 ; white, 26,000.

There was appreciable swelling of the right parotid.

Jan. 1st and 2d : Numerous tarry stools and dejections of fluid blood. The patient had several epileptiform seizures.

Jan. 3d : Death occurred following copious melæna.

The temperature, except for a few hours following the collapse on the 27th when it fell to 99°, was continuously above normal (100°–102°).

Autopsy : Anatomical diagnosis: Chronic endocarditis, adhesive peritonitis, ulcer of duodenum.

Lungs : A few light adhesions over base of both lungs.

Heart : The mitral curtains are thickened and show a few small vegetations.

Liver : Consistence, firm ; surface smooth, section a little pale ; weight forty ounces.

Spleen : Surface normal, normal in consistence and on section ; weight eight ounces.

Gall Bladder : Full of very dark bile.

Kidneys : Normal in appearance and on section ; micro. ex., practically negative.

Adrenals : Normal in appearance and on section.

Pancreas : Adherent at head to duodenum, otherwise normal in appearance and on section.

Stomach : The mucosa is normal in appearance.

Peritoneum : There are very firm adhesions between the diaphragm and upper surface of liver. There are also firm adhesions between the duodenum and under surface of liver.

Duodenum : There is an oval ulcer one and one quarter inches in diameter, the upper edge of which is a half-inch below the pyloric ring. Its edges are abrupt, and the floor is formed by the adherent head of the pancreas. In the floor of the ulcer is seen an opening into an eroded artery of large size.

Intestines : Otherwise normal throughout their extent.

Case 5.— Male, æt. twenty-five. Bartender. Admitted to hospital August 5, 1893. Service of Dr. Thompson.

The patient was brought to the hospital in an ambulance, with the sole history of two days' alcoholism and bowel disturbance (?). It later was learned that he had been at work on the day previous to admission, and had suffered for an indefinite period from an alcoholic gastritis (?). The patient had a convulsion in the reception room and was immediately sent to the wards.

Physical Examination : The patient is in a state of profound collapse. Heart action very rapid and feeble, pulse almost imperceptible. Temperature (rectal), 104.8°.

The abdomen is tense and painful.

Further examination was impossible as the patient was moribund and died a few moments later.

Autopsy: Anatomical diagnosis : Chronic endocarditis, perforating ulcer of the duodenum, septic peritonitis.

Heart : The mitral valve is thickened and presents a few small vegetations. The other valves are normal. Muscle normal in appearance and on section. Weight 11½ ounces.

Lungs : Normal, beyond a few old adhesions of right pleura and a moderate degree of congestion of both lungs.

Peritoneal Cavity : Contains 1000 c. c. of sero-pus, with a considerable amount of fibrino-purulent exudate.

Stomach : The mucous membrane appears to be slightly thickened, otherwise normal.

Intestines : Duodenum : there is a perforating ulcer one half inch in diameter, on the anterior wall, at the junction of the duodenum with the pylorus. Intestines otherwise normal.

Liver : Surface smooth, color pale, consistence firm. On section very pale and fatty. Weight 104 ounces.

Gall Bladder : Contains a small amount of brown bile.

Spleen : Surface normal ; on section, normal.

Pancreas : Normal.

Kidneys : Left, weight 5½ ounces. Right, weight 6 ounces. Capsules, non-adherent. Surface smooth. On section, striæ a little indistinct. Histological examination : slight degeneration of the cells of the convoluted tubes.

Brain : Membranes normal, substance normal.

Case 6.— Male, æt. thirty-four. Canadian. Tradesman. Admitted to St. Luke's Hospital April 9, 1885. Service of Dr. Wheelock.

Family History : Unimportant.

Previous History : Never any acute illness, and never any rheumatic symptoms. Formerly, a moderate alcoholic habit. Ten years ago, a venereal sore and suppurating buboes. The patient denies other specific history.

Present Illness : Insidious onset : the patient worked until three months ago, but for some time previous had suffered from occasional faintness, dyspnœa and palpitation on slight exertion, and slight swelling of the feet. From time to time there were attacks of almost incessant vomiting for a period of twenty-four hours, but there was no blood in the vomited matter (uræmic ?). Beyond this symptom, there was apparently an absence of gastric disturbance. He has been troubled with frequent micturition and occasionally there has been difficulty in passing urine. In the past three weeks he has expectorated frequently a small amount of bright-red blood. There has been a progressive loss of weight and strength.

On Admission : Physical Examination : *Heart :* The apex is one and one half inches to the left of the mid-clavicular line. There is a double rasping murmur over the aortic area ; the diastolic murmur is also heard at the apex. There is also a systolic murmur over the mitral area, and accentuation of the pulmonic second sound.

Lungs : There are the physical signs of fluid in the right pleural cavity. There is no ascites, but a moderate general subcutaneous œdema. Pulse 92 (Corrigan's), temperature normal.

Urine : Sp. gr. 1.008, albumin a trace, no sugar, the examination shows a considerable amount of pus.

From the date of admission until April 30th, when death occurred, there were frequent hæmatemeses, some of them being of considerable amount. No blood was passed by the bowel. The anasarca gradually increased, signs of rapidly accumulating fluid in the peritoneal cavity were present ; but at no time were there any symptoms of collapse, and death occurred apparently as the result of the cardiac lesion.

Autopsy : Anatomical diagnosis : Perforating duodenal ulcer ; general peritonitis ;

cardiac hypertrophy and dilatation; aortic insufficiency; chronic diffuse nephritis; renal calculus.

Heart: Left ventricle hypertrophied and dilated, muscle pale, soft, and contains a good deal of fat. Two segments of the aortic valve are agglutinated, forming an imperfect segment. There is stenosis with insufficiency. Weight, 16 ounces.

The mitral valve is normal with the exception of a calcareous deposit at its base. The other valves are normal.

Lungs: The right pleural cavity contains 24 ounces of clear serum. Both lungs contain a number of infarcts.

Peritoneal Cavity: Contains 136 ounces of bile-stained serum, and considerable recent plastic exudate.

Stomach: Normal.

Duodenum: There are two ulcers in the wall of the duodenum, one irregularly oval in shape and involving the entire thickness of the mucosa, the second circular in contour and communicating with the peritoneal cavity by an opening $\frac{1}{8}$ inch in diameter.

Kidneys: Section shows the presence of a chronic diffuse nephritis. There is a calculus in the pelvis of one kidney.

Case 7.—Male, æt. thirty-four.

Family History: Unimportant.

Previous History: There is no alcoholic or specific history. There has been an abuse of tobacco at different periods from boyhood. Digestive disturbances of unusual severity have been present at various times from early youth. They have consisted of attacks of acute epigastric pain increased by the ingestion of food and always partially or wholly relieved by vomiting. The attacks were of varied duration. Periods of several months of suffering would be followed by intervals of relief of a corresponding or longer duration. Neither hæmatemesis nor melæna occurred at any time. An inordinate craving for "sweets" and an indulgence of the taste has existed for many years. For six months previous to the beginning of his present illness comparative good health has been enjoyed. A pound of candy has been consumed daily by the patient for the latter three months of this period. A month before the patient came under my observation, severe epigastric pain began to be experienced. The pain was constant, being neither increased nor diminished by the ingestion of food. Gradually vomiting followed directly the taking of food of any kind. Hæmatemesis never occurred and blood in the stools was never detected.

The patient came under my care suffering from the symptoms described. He was greatly emaciated. Physical examination of the various organs failed to elicit any evidence of disease. The urinary examination was negative. Although severe epigastric pain was complained of, a painful pressure-point was absent and general epigastric sensitiveness was not marked. The epigastric pain was constantly present and apparently was little affected by the ingestion of food, which at first the patient was able to take, although vomiting was frequent. Speedily, vomiting occurred directly on the taking of food of any kind. Lavage was employed without effect. Finally it was impossible to feed the patient otherwise than by the rectum. During the period of rectal alimentation the pain persisted but was somewhat ameliorated. Hæmatemesis never occurred, and although the stools were systematically and carefully examined the presence of blood was never detected. There was continuous and rapid loss of strength and weight. The patient refused to permit any attempt to relieve his condition by operation. The emaciation finally became extreme and death resulted from inanition at the end of the fourth week.

Only a partial autopsy was permitted. The stomach was examined in situ, and no open ulcer was discovered. From the clinical history, it is probable that cicatrices of healed ulcers existed. Dilatation of any marked degree was absent. The duodenum was firmly

adherent to the under surface of the liver in such a manner as to cause a partial stenosis of its first portion. On the anterior wall about an inch from the pylorus there was a round ulcer, an inch and a half in diameter, with thickened, indurated, and abrupt edges, the floor being formed by the serous coat.

A further examination was not permitted.

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ON GROWTH AND OVERGROWTH AND ON THE RELATIONSHIP BETWEEN CELL DIFFERENTIATION AND PROLIFERATIVE CAPACITY; ITS BEARING UPON THE REGENERATION OF TISSUES AND THE DEVELOPMENT OF TUMORS.

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THERE are in medicine and other sciences not a few beliefs and ideas which we have acquired we know not how, and which in general we no more think of discussing than we do, for example, the subject of good manners. Thus, just as it is difficult if not impossible for us to state how or where we gained any single article of our code of personal ethics, so it is with certain of these general ideas in medicine; and while we have never discussed these beliefs we feel individually assured that they are the current ideas of other workers along the same lines.

One of these tacit beliefs or comprehensions relates to the subject of the growth of tissues. Asked offhand we should assuredly, each one of us, state as his familiar belief that muscle arises from muscle, epithelium from epithelium, nerve cell from nerve cell, and so on, and the mental picture which we form of the process of growth is, I fancy (though here, of course, I speak under correction), that the fully formed epithelial cell undergoes mitosis and divides into two, and so with the cells of other tissues. Yet if we think a little longer and recall what we have actually seen under the microscope, this mental picture is seen to be incorrect or, at least, imperfect. Thinking recently over this matter it has been impressed upon me that if we obtain a correct idea of what occurs during the process of growth of tissues, we not only realize that there is a very broad biological law underlying this process, from the earliest stages of the embryo upwards, but, further, applying this biological law to the subject of pathology, we gain a deeper and

a fuller comprehension of certain matters bearing upon regeneration and degeneration of tissues, and, what is more, bearing very directly upon certain of the phenomena of tumor growth.

While the individual, formed as he is of a marvellous complex of various tissues, is a reproduction of the tissues and organs present in his paternal and maternal ancestors, he is the outcome and development not of the combined fully developed tissues, nor again of any one highly differentiated cell or cell-compound, but of a single undifferentiated cell,— the fertilized ovum,— a cell neither of the component parts of which, ovum or spermatozoön, has ever, through the whole course of the ages, been derived from other than similar undifferentiated cells; or, in other words, these apparently simple germ cells are capable of giving rise to the whole series of cells forming the whole mass of tissues from the simplest connective up to the most highly differentiated nerve tissue.[1] If we study the process of the development of the embryo, whether in plant or animal life, we see again a somewhat similar phenomenon.

From a very early period in such growth we recognize that certain cells alone appear to be actively dividing and to be actively proliferative, whereas other cells, the products of these, while they take on characteristic appearances, do not thus divide. Very early indeed in the developing embryo we recognize this existence of what may be termed "mother cells,"— cells which themselves remaining embryonic in type give rise by division to other cells which assume more highly differentiated characters. And this is true not only of animal organisms, but still more obviously of plants: we have but, with Sachs, to study the growing points of plants to realize the existence of these "mother cells."

Now when we pass to the fully formed individual, we must still recognize this fact, which we are apt to pass over, namely, that the specific cells, if I may so term them, of the different tissues — that is, the highly differentiated and characteristic cells of those tissues — do not themselves give rise directly, and by division, to other specific cells, but that in each tissue there are these more or less undifferentiated mother cells, which more especially have the power of proliferating, and it is the daughter cells which assume the full specific properties in

[1] It is possible that while the cell-body of these germ cells is relatively undifferentiated, the nucleus is peculiarly highly elaborated. We have, indeed, not a few indications that this is the case. Thus it may be that nuclear and cytoplasmic differentiation, in germ and "mother" cells, and in specific tissue and "daughter" cells respectively, are in inverse ratio. We have not as yet reached a point at which any definite statement can be made in this connection. Throughout this article I refer to cytoplasmic and not nuclear differentiation.

the different tissues, or if, as would seem to be the case in some instances, the active cells of certain not very highly differentiated tissues themselves proliferate, they only do this *after a preliminary reversion* to a "mother cell," or more embryonic type.

Let me here give a few examples: Immediately I mention them every one will realize that these are facts thoroughly familiar. One of the simplest cases is that of the skin; in this it is the lowest layer of the epidermis, the Malpighian layer, whose cells undergo active proliferation, and this constantly, throughout the whole period of life, and the cells which are most active in the process are those of the very lowest layer — cells which are of a permanently embryonic type.

After injury, it is true, we may come across occasional mitotic increase, more especially in the lower animals, in the region of the prickle cells, but, when we do, we see that it is accompanied by reversion of the cell to a simpler type: the prickles or bridges disappear, the cell loses its connection with the neighboring cells, and passes back to a simpler stage prior to multiplication.

In bone the case is the same. Production of bone tissue is *not* brought about by multiplication of the pre-existing and characteristic bone cells, but by osteoblastic tissue; or, in other words, by the mother cells of bone lying in the immediate neighborhood of the vessels of the inner layer of the periosteum, or again coursing through the Haversian canals and between the laminæ. When through injury or otherwise a stimulation is afforded, it is these mother cells which, applying themselves to the pre-existing bone, multiply, and there, governing the spaces around them, lead to the deposit of a calcareous or bony matrix and gradually assume all the characters of typical bone corpuscles.

From the variability of the structure of the glandular organs it is difficult to lay down any general rule with regard to regeneration of glandular epithelium, so that it must be confessed that, partly on account of this variability, partly on account of the fact that the different observers upon glandular regeneration have not had this conception of mother cells before them in their studies, such glandular epithelium in the present state of our knowledge affords the least satisfactory demonstration of the principle. There are, however, so many instances concerning which we have positive information, as to render it more than probable that throughout the same principle is in action.

Of the mother cells of the type seen in the Malpighian layer of the epidermis, several instances may be called to mind — the mother cells of the testicular epithelium and (though differing somewhat in plan) of the ovarian follicles, of the sebaceous and, apparently, from Tornier's studies, the mammary glands. In both of the latter organs there is,

during activity, a giving off of cells, and mitosis occurs especially in the deeper, more external layers.

In this connection should, I think, be mentioned the columnar and ciliated epithelia of mucous membranes. It is remarkable how one histological text-book after another figures such columnar epithelium as formed of a palisade of fully developed columnar cells in regular series situated upon the basement membrane, without indicating or even referring to the presence of embryonal "mother cells" lying between the bases of the fully differentiated columnar cells. Certainly under pathological conditions one is impressed by the fact that these basal cells are present, and that there is a development of new cells from beneath to take the place of the degenerating fully formed cells. If in the simplest acinous glands it is difficult to recognize such permanent mother cells (indeed they are probably absent in the adult), here at least they are present.

In the lymph glands we have another arrangement. In them the mother cells are collected in the form of small nodes in the centres of the individual follicles. A somewhat similar arrangement of foci of embryonal cells is met with in the thyroid, while in the pancreas, Langerhans's bodies or "cell accumulations" would seem to be of a similar nature, although this is not as yet absolutely determined.

Closely allied to this condition is what is seen in the lung and again in the serous coat of the peritoneum (though neither of these, it is true, comes strictly under the category of gland structures). In the former, certain more embryonic cells persist, especially at the angles of junction of the alveoli, and these are generally regarded as centres for the normal proliferation and regeneration of the alveolar epithelium. On the peritoneal surface also there are found clumps of smaller cells capable of active proliferation. With regard to both these cell layers, the flattened — differentiated — cells are liable, under irritation, to revert to the fuller, more rounded embryonic condition and then undergo mitosis and proliferation.

Judging from the fact that mitoses are not observed in the fundal portions of several acinous glands, while they are frequent in the neck regions of the same, it would seem at least probable that mother cells, or cells capable of reverting to the mother-cell type, are situated in the latter area. But generally in acinous glands of simple type there is, as Bizzozero has pointed out, little evidence of continued proliferation under normal conditions, once they are fully formed. In connection with these, it is that more exact studies are requisite; certainly in some, *e. g.*, in the kidney tubules, slight continued irritation leads to the differentiated cells undergoing proliferation, but at the same time

these cells are seen to revert to a simpler type. Thus, while confessing that it is not as yet possible to demonstrate in every case that either mother cells are present or that for regeneration the differentiated cells revert to a more embryonic type, the number of examples that can be brought forward, where either one or other of these processes is seen to occur, is so large that we may assume that a general principle obtains in connection with all.

Passing now to a tissue of another type, namely muscle, the process of regeneration, as pointed out by Barfurth, varies largely according to the age of the individual. Originally the muscle fibres, it is needless to say, are recognized as arising from a series of cells known as sarcoblasts, relatively large cells with abundant protoplasm, the nuclei of which tend to proliferate, and along one aspect of, or generally around, the periphery there is gradually developed first a longitudinal fibrillation, and then transverse striation. In young larval forms of amphibia, for example, after injury such sarcoblasts may be seen to be present, to undergo multiplication, and to give rise to typical muscle fibres; here, therefore, very clearly there are mother cells. The existence of such in the adult is still a matter of dispute; for in the older individuals two processes have more especially been recognized: one, that in which the fully formed muscle fibres become the seat of an active nuclear proliferation, and then in certain areas of the fibres the striation, more especially toward the ends, or laterally, becomes indistinct and little buds containing nuclei are given off, which become the precursors of the new growth. Or secondly, as pointed out first by Weismann, there may be recognized, even in the perfectly normal muscle of adult animals, certain occasional spindle-like bodies, consisting of short striated muscle fibres, characterized by very abundant nuclei and, according to Kölliker and others, these bodies represent centres from which by longitudinal division new fibres are given off. Even in this case it will be seen that these bodies represent a lower, imperfect stage of differentiation, and the richly nucleated mass may strictly be regarded as a "mother cell," or node of "mother cells."

Thus from a study of the process of regeneration as seen in the different tissues in man and vertebrates generally, it is possible to lay down the following laws:

1. **The fully differentiated cells of a tissue proper never arise from cells that are themselves fully differentiated.**

2. **Under the *normal* conditions of growth and during physiological regeneration, the fully differentiated cells would seem in nearly every case to be developed from "mother cells"—undifferentiated or but partially differentiated cells, which**

themselves throughout the term of life of the individual never attain to the full differentiation peculiar to the tissue to which they belong and which indeed they produce. For these mother cells by division give origin to the daughter cells, and it is the daughter cells which attain full differentiation and form the specific cells of the tissue. More rarely the functional cells themselves, by reversion to a more embryonal type, take on the properties of mother cells.

3. Under *abnormal* conditions, the fully differentiated functioning cells of certain tissues are capable of proliferation and giving rise to cells of like nature, but this is only after a preliminary reversion to a simpler, more embryonic type. The fully differentiated cell as such is incapable of proliferation.

4. Or, otherwise, the energy stored up by the cell may be expended in one of two directions, but not in both — either in functional activity or in preparation for proliferation, and, the structure of a cell being the expression of the activity of that cell, the expenditure of energy in either direction is attended by corresponding morphological or structural differences in the cell.

5. The more highly differentiated a cell, the more highly elaborated its structure, the less the ease with which it reverts, and the less the liability to reversion to a simpler reproductive form; the simpler the cell, the greater the ease and the greater the liability to such reversion. It is thus possible to conceive, at the one extreme, cells so simple in function and in structure that functional or reproductive activity may be called into play indifferently, without *recognizable* preliminary structural alteration, and, at the other, cells so highly differentiated that the capacity for proliferation has become entirely lost.

The law with its corollaries here given does not so far appear to have obtained general recognition. Certainly I fail to find it explicitly referred to by writers upon morbid anatomy, and had it been currently accepted by biologists it would ere this have become part and parcel of pathological teaching. It is only after some little search that I find its main paragraph laid down by Kölliker in 1885, and that only, as it were, in passing, in an article not dealing directly with regeneration, but dealing with the part played by the nucleus in inheritance. In that article Kölliker states: "In all cases in which an organ or a tissue is capable of regenerating itself, it must contain elements of an embryonal character, or at least such elements as can take on that character." And again, in the same article: "These cells then are dependent upon the same law, as, in the embryo, is the formation of organs."[1] But in the general portion of his great *Handbuch der Gewebelehre des Menschen*, published in 1889, I cannot find that he again

[1] Kölliker: "In allen Fällen, in denen ein Organ oder ein Gewebe fähig ist sich wieder zu erzeugen, muss dasselbe Elemente von embryonalem Charakter enthalten oder wenigstens solche, die diesen Charakter anzunehmen im Stande sind." "Diese Zellen bedingen dann nach denselben Gesetzen, wie beim Embryo, die Organgestaltung."—"Die Bedeutung der Zellenkerne für die Vorgänge der Vererbung," *Zeitschrift f. wiss. Zool.*, vol. xlii., 1885. (Quoted by Barfurth, "Zur Regeneration der Gewebe," *Arch. f. mikrose. Anatomie.*, xxxvii., 1891, p. 424.

refers to this principle, hence it would appear that he did not realize its wide application.

Thus, although the law when once grasped is simple, almost to the verge of being a truism, it merits being stated for once in as clear terms as possible that its high importance may be fully recognized.

Applying now these considerations to certain pathological conditions, it is in the first place necessary to say but little concerning pathological regeneration and repair, for I have already applied some of the facts gained from a study of such regeneration in the development of my argument. Thus much, however, may be said, that the more we study the regeneration of various tissues, the more we see that the phenomena of the process fall into place in consonance with the law above laid down. The cells of a regenerated tissue are derived from the pre-existing cells (and mother cells) of that particular tissue, and the more highly differentiated the tissue the less is its capacity for regeneration.

Of all cells, those which are the most highly differentiated, namely the nerve cells, show the least capacity for proliferative regeneration. There is a remarkable lack of evidence of fully developed nerve cells having been observed in the process of mitosis, and, corresponding with this lack, when we consider the histological basis of motor, sensory, and mental acts, it is *a priori* hard, if not impossible, to conceive a given cell in any of the higher nerve centres undergoing proliferation without there being simultaneous arrest, not to say loss, of function. Everything indicates that of all the cells of the body the neurons are those endowed with the longest life period.

During fœtal or larval life, prior to the attaining of full function, such proliferation has been recognized (Barfurth), or, otherwise, the incompletely differentiated nerve cells may take part in the regenerative process; though it may be considered an open question whether the new development of neurons in experiments upon the spinal cord of the larval newt and other amphibia is the result of proliferation of developed neurons or of mother cells.

That such mother cells must exist even in the human nervous system has been forcibly brought home to me by a study of certain sections of the spinal cord to which Dr. Shirres, working in my laboratory at the Royal Victoria Hospital, recently called my attention.

These sections are from a case of porencephalus in which apparently, from all indications, the atrophy and disappearance of practically the whole motor area in the left hemisphere dated from the last month or two of fœtal life. Despite this practically complete loss of the entire motor area on the left side, the resulting evidences of paralysis and

disturbance of function were little more than a relatively slight want of development of the bones and muscles of the right side of the body, together with paresis of the musculature of the right hand. In the cord the difference between the two motor tracts is most marked, there is entire absence of the crossed pyramidal tract on the left side, of the direct pyramidal on the right, and, corresponding with this, there is a most marked atrophy of those groups of cells towards the periphery of the anterior horn of the grey matter on the right side which everything indicates as being connected with the crossed pyramidal tract and being what I may term the spinal motor centres for the upper extremity. But in sections in which this atrophy is most marked there is a distinct increase in the number of ganglion cells in the deeper and more central areas of the grey matter; here the number of cells is much above the normal, the cells are large and more numerous than those of the left side in the same section.

For our present purposes it is clear that these abundant cells cannot be regarded as due to proliferation of the atrophied cells — their relative position forbids such an assumption. Their existence, however, is strongly in favor of the view that they have been derived from mother cells in the grey substance.

The regeneration of nerve fibres is another matter. So far as regards the axones, or axis-cylinder processes themselves, it is strictly comparable with the new growth of the cell substance of the amœba when a part of that cell substance has been removed without direct injury to the nucleus. As regards the investing sheath of the medullated nerve fibre, regeneration here is in strict accordance with the third law there; is a preliminary period of reversion; the formed substance (myelin), governed or elaborated by the cell, breaks down, the nucleus becomes more prominent and surrounded by an increased amount of protoplasm, and then in place of a single nucleus of a node of Ranvier one gets numerous nuclei, and the new cells evidently surround the newly developed axis cylinder as it grows downwards from the seat of injury.

I have already referred to the regeneration of muscle, but if now we pass to the other extreme and consider what happens in the simplest of all tissues, namely, white fibrous connective tissue, we find that even here for pathological regeneration to occur there is a preliminary stage of swelling of the connective-tissue cells, the nuclei become larger and more prominent, the surrounding protoplasm becomes increased in amount, the surrounding fibrils tend to disappear, and now, only after this reversion to a simpler, more embryonic condition, do we have proliferation and the subsequent building up of new connective tissue.

To enter here into a discussion as to whether connective tissue can

be formed from wandering cells, and whether any special form of wandering cell is capable of taking part in such new formation, would be imperative if I were dealing more especially with the law that tissues arise from pre-existing specific tissue cells; here rather, dealing with the process and the law of growth, with the law of mother cells, if I may so term it, the very limits to which this article is confined by the editors must be an excuse for not dealing with this subject.

A few words, however, must be said with regard to metaplasia in general, for the law of reversion to a more embryonic type is distinctly in evidence throughout, and indeed helps us to understand how, for example, a columnar-celled epithelium can be converted into a squamous-celled epithelium and one form of connective tissue into another; or, otherwise, metaplasia is never direct, but is only brought about by preliminary reversion to a more embryonic type, or, where mother cells are present, by the modified development of cells derived from the mother cells, the influence of environment altering the character of those daughter cells during the period of growth.

If the principle be correct that the more highly differentiated a cell, the less its capacity for proliferation; or, in other words, the more the daughter cells depart from the type of the mother cells, the less their proliferative capacity, then the converse would seem to hold — that *the more the daughter cells in a given tissue retain the characteristics of the mother cells, the greater their proliferative capacity.* Or — to continue the argument — if through any cause the daughter cells in a tissue do not attain full specific differentiation, then they are peculiarly liable to proliferate and function, not as specific cells, but as mother cells. And further, as Bizzozero has pointed out, tissues in which the cellular elements exhibit frequent mitosis are those which more especially are liable to be the seat of excessive growth and tumor formation.

I am far from saying that this is the one principle concerned in the development of the blastomata or tumors proper, but I would urge that it is one important principle. It is outside the limits of this article to consider what is the essential stimulus, or what are the stimuli, leading to neoplastic growth; accepting, however, for the moment, that, whether temporarily or permanently, some stimulus or stimuli be in action, at least we gain a comprehension of why in the first place histioid tumors (in which one or other tissue is more or less faithfully reproduced) are essentially benign; and in the second place why malignancy and excessive cell proliferation (which is the essence of malignancy), go hand-in-hand with tumor formation of a pronounced embryonic type.

The failure to recognize the normal existence of mother cells in those tissues which are prone to tumor formation is very obvious in reading over Cohnheim's chapter upon tumors and in following the course of the argument which led him to conclude that tumors arise from latent superfluous embryonal tissue either lying within a tissue of the same nature or heterotopic.

While we must admit the existence of embryonal rests or inclusions in order to explain the development of such tumors as, for example, myomata of the kidney and primary cancerous growths developing in bone, the permanent existence of these mother cells as the normal constituent of the tissues throughout life is wholly adequate to explain most benign and malignant tumors. To this larger conception of what is " embryonal tissue " we must, I think, inevitably come, and with Senn and others we must regard tumors as products of tissue proliferation of embryonic cells of either congenital or post-natal origin, even though we at present continue unable to state definitely what it is that immediately induces these cells to undertake excessive growth. Granting this, that in tumors we have aberrant tissue growth, and that that tissue growth is due to certain cells assuming excessive proliferative properties wholly outside the needs of the economy, then tumors are to be considered as being derived :

1. From embryonic cell " rests " which have for a shorter or longer period remained latent in one or other tissue and then have taken upon themselves a rapid proliferation leading to tumor formation.

2. From the mother cells of a tissue which, remaining undifferentiated, but capable of active proliferations throughout life, now assume excessive proliferative powers, their daughter cells retaining to a greater or less extent the characters and the properties of the mother cells.

3. From differentiated cells which reverting to a simpler, more embryonic type, with this reversion gain the capacity for active and excessive proliferation.

Possibly, I may add, the tendency to the development of glandular cancer in later life bears some relationship to the reversion and degeneration of gland cells at this period. As the tissues become exhausted, the more highly differentiated cells tend to become structurally simpler, revert, that is to say, to a simpler type, and with this simplification of structure accompanying atrophy there may be, I would suggest, a greater liability for those cells to assume proliferative powers, along the lines already laid down.

I do but suggest this. Space does not permit that I should here discuss the age incidence of one or other form of neoplastic growth. I trust, however, that in these few pages a sufficiency of facts has been brought forward to indicate that this conception of " mother cells," or

as the botanists term them, "Cambium cells," and of potential mother cells is of no little value for a comprehension of the etiology of tumor growth as of cell proliferation in general, and to show that fuller studies along these lines promise to advance our knowledge of that etiology very materially.

SEPTIC ENDOCARDITIS.

By SAMUEL S. ADAMS, A.M., M.D.,

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THE interest manifested by my colleagues of the Children's Hospital in a case of septic endocarditis on my service in that institution, the details of which will be given hereafter, induced me to collate the cases in children fourteen years of age and under, as well as to review the literature of endocarditis in its relation to infection as the causative element. The difficulties encountered in this attempt were often embarrassing, and at times appalling, because (1) the subhead "Endocarditis," as indexed at the Library of the Surgeon-General's office, is lost under the headings "Heart (inflammation of)," "Heart (disease in children)," etc.; (2) a number of the cases here collated were not indexed, and in many instances were traced from allusions to them, found by collateral reading; and (3) owing to the confusion in the classification of the varieties of endocarditis, the greatest liberality being shown in the descriptive adjective; on the one hand the terms "simple" or "benign," and on the other "ulcerative," "malignant," "infective," "diphtheritic," "septic," "fungating," and "mycotic," being subject only to the license of the author or reporter.

The effort has been made to collate only such cases as may be considered "infective" from the specific statement of the author, or from the characteristic clinical picture by which the disease is more frequently diagnosticated than from either bacteriological or post-mortem findings. It is not claimed that every published case is included in this list, for in all probability there are some recorded in text-books or incidentally mentioned in other works which have escaped us, but it is certain that all indexed cases, as well as those which were accidentally discovered, are here tabulated.

Unless we attribute the small number of cases reported to failures in diagnosis, septic endocarditis in children may be considered to be a rare affection. Donkin says it is not frequent in childhood, and has no clinical peculiarity at this period. He saw only three probable cases,

which were suspected during life, only one having been submitted to post-mortem examination, which alone gives positive evidence of this disease. It is sometimes found post-mortem where it is not suspected, but it is still more often suspected where it is not found.

Gorwitz, on the other hand, draws the following conclusions: (1) By the side of the classical infectious endocarditis, we place, in the cases described, a form as yet little known. (2) This form is rather peculiar to infants. (3) The beginning is insidious, the progress slow, the heart is little affected, as also the other organs. (4) The infection dominates the scene, leaving in the second plane the circulatory apparatus. (5) The prognosis, while grave, is not [necessarily] fatal; it is subordinate to the degree of infection. (6) The invalid may recover, but the affection leaves, as a trace of its passage, an organic lesion of the heart.

Osler says that in 209 cases of endocarditis which he has studied, there were but three under ten years of age. Whittaker says: "It is rare in infancy, but begins to show itself with the occurrence of various infections." The records of the Children's Hospital at Melbourne furnish but one case in ten years. Davies finds but one case similar to his own in the Transactions of the Pathological Society (London); it is that of a girl one year old, under the care of Dr. Sharkey. This case we have been unable to find elsewhere.

The earliest case, that recorded by Kirkes, who first recognized and described the disease, was in a boy aged fourteen years. Only a single case appears in the records of the London Hospital for Sick Children during the two decades ending in 1890; and my case is the only one treated in the Children's Hospital, D. C., since its organization in 1870.

Kanthack and Tickell have studied 84 cases in St. Bartholomew's Hospital from January, 1890, to March, 1897. The following were found in children:

By Age — 1 to 5 years = 1 M. — 0 F.
5 to 10 " = 2 " — 2 "
10 to 15 " = 2 " — 4 "

Of these 84 cases, 60.7% were males, 39.29% females.

62.7% of males between twenty and forty years.
51.5% of females between ten and thirty years.
33.3% of females between fifteen and twenty-five years.[1]

In connection with the statistics given by Kanthack and Tickell, those of Thiele are interesting. Between 1873 and 1887, 4979 necropsies were made in the Pathologic Institute at Kiel. Of these, endo-

[1] This note added lest the relative predominance of females in list lead to erroneous conclusions.

carditis was found in 373 cases, 168 (45 %) in females, 209 (56 %) in males, and the sex not stated in 1.

Under	1 year	5	M.,	6	F.	Total 11.
1– 9	years	10	"	8	"	" 18.
10–19	"	14	"	12	"	" 26.

Fresh hemorrhages, old hemorrhages, infarcts, and embolic abscesses were found in the brain, retina, lungs, liver, spleen, and kidneys.

While it is generally admitted that septic endocarditis is a specific affection, nevertheless there have been many pathologists, bacteriologists, and clinicians unwilling to accept it as such. Lazarus-Barlow contends that, in spite of the fact that physicians recognize two varieties of acute endocarditis (simple and malignant), and that they differ in their clinical pictures, the difference between simple and septic endocarditis is one of degree, dependent upon the virulence of the micro-organisms or their toxins. He admits that micro-organisms are almost invariably found in the vegetations on the valves in infective endocarditis, but asserts that they are frequently found in the *so-called* simple variety, and that the same micro-organisms are present in both conditions. According to his interpretation, the micro-organisms act as irritants, producing only inflammation in simple, but inflammation plus necrosis in septic, endocarditis. His explanation of the difference in the pathological processes produced by the emboli which are broken off from the vegetations and carried in the blood-currents is that such emboli are *aseptic* in simple, and *septic* in infective, endocarditis. The effects also of the two varieties upon the heart itself are merely differences in degree; both forms produce changes in the myocardium, though the ulcerative, from the effects of its toxins, produces greater impairment of function of both heart muscle and valve.

Weichselbaum claims that there is no essential difference, either histologically or bacteriologically, between the two forms of endocarditis; that the same species of bacteria are found in both; and that in those cases in which bacteria have not been found they have either escaped detection or have died.

The trend of opinion to-day seems to accord with the views of Lazarus-Barlow and Weichselbaum that the two conditions differ in degree but not in kind; and one author suggests that the burden of proof falls upon him who would present a case of *non*-infective endocarditis.

Rosenbach, in 1878, induced acute endocarditis, experimentally, in animals by injuring the heart-valves with a sound passed into the carotid; and, in 1885, Wyssokowitsch and Orth induced it by injecting

pyogenic organisms into the circulation after the heart-valves had been injured by Rosenbach's method.

A study of the experimental work of Netter, Ribbert, Fränkel and Sänger, Prudden, Weichselbaum, Stern and Hirschler, Viti, and others shows that true endocarditis can be produced in animals by injecting into the veins any one of the numerous micro-organisms, provided the heart-valves are diseased, although Netter and Ribbert think a previous lesion is not essential; and Dreschfeld and others have succeeded in reproducing the disease in animals without previously injuring the valves.

According to Wright and Stokes: "The first extensive study, however, of the etiology of acute endocarditis in man is that of Fränkel and Sänger, published in 1886. They studied eleven cases of verrucose and one of ulcerative endocarditis, and in nine of them found bacteria in the cardiac lesions, the staphylococcus pyogenes aureus being present in six." Netter found the micrococcus lanceolatus in nine cases of acute endocarditis associated with croupous pneumonia. Of the twenty-nine cases of Weichselbaum, the micrococcus lanceolatus was found in seven, the streptococcus in six, the staphylococcus pyogenes aureus in two; and in six, various unusual bacteria; while in eight cases no micro-organisms were found. In ten cases, Wright and Stokes examined the valvular vegetations and ulcerated tissues of the heart and found the micrococcus lanceolatus in seven, one being accompanied by the bacillus diphtheriæ; the streptococcus in one; streptococcus and staphylococcus in one; and unknown in one.

Dessy found in thirty-six cases of endocarditis (three being ulcerative and showing pneumococci) bacteria in thirty-four, viz.: pneumococcus in thirteen, streptococcus in twelve, staphylococcus in three, and other species in six. He considers the pneumococcus and streptococcus, either alone or associated with other forms, the cause. Lancereaux says that when infective endocarditis is found in connection with malarial poisoning, a frequent occurrence in Africa, its seat of predilection is in the aortic valves. Dessy informs us that the pneumococcus selects the aortic valve, and the streptococcus the mitral. Again, Raue explains "the almost invariable occurrence of endocarditis upon the left side of the heart, during extrauterine life, and a similar selective affinity for the right heart in fœtal endocarditis," upon the ground that "endocarditis is an infectious condition" and "the germs which excite inflammatory reaction in the endocardium can thrive only in a medium of arterial [*i. e.*, freshly oxygenated] blood"—an idea perhaps indicated by Giraudi.

Dreschfeld summarizes the results obtained by different observers as follows:

1. That microbes were found in nearly all cases of infective endocarditis, regardless of ulceration.

2. That most observers found but one organism in their cases; a few, however, found two or more.

3. That the same organism was not found in all. In some the organism found was also met with in other infectious diseases; and in others the organism had not been found in other diseases.

4. In most cases, the organism found in the diseased valve also invaded the infarcts and metastatic abscesses.

5. The streptococcus, the staphylococcus, and the pneumococcus are the organisms most frequently found.

D'Astros comments on the difficulty of demonstrating the several steps of these infections during life; Achalme, for instance, in a case of endocarditis complicating erysipelas, having failed to find the streptococcus during life.

The disease may be considered rarely primary. Osler states that it is engrafted upon a pre-existing valvular lesion in seventy-five per cent. of cases; hence the micro-organism invades the broken endocardium.

Dreschfeld says we may distinguish the following types of infectious endocarditis: (*a*) primary; (*b*) as a complication of septic disease; (*c*) as a complication of pneumonia or meningitis, and due to the diplococcus pneumoniæ; (*d*) as a mixed infection, due to septic organisms secondary to the acute infectious fevers, or secondary to rheumatic endocarditis or sclerotic condition of the valves.

Lazarus-Barlow says in many cases of pyemia a primary focus of suppuration is found, but this is not invariably the case, to account for the metastatic abscesses. In such cases cardiac symptoms develop and the necropsy reveals ulcerations on the valves. He admits in such cases the lesion in the endocardium as the immediate antecedent cause of the abscesses, because the pyogenetic organisms have been found there. "Where this is the case, association of the secondary abscess with embolism of particles of the septic vegetations on the valves is easy, especially in view of the fact that in these cases the metastatic abscesses are most commonly found in the spleen and kidneys, regions in which simple embolism and infarction are especially common in valvular disease of the ordinary kind." It is not easy to explain the valvular disease, which in a majority of cases antedates the ulcerative process. The micro-organisms must gain access to the blood in order to produce their harmful effects. In all cases in which the micro-organism is localized, and there are associated with it valve-lesions

and metastatic abscesses, the relation between cause and effect is apparent. The blood is invaded from a primary focus situated in the tissues. He assumes, therefore, the pre-existence of a primary superficial focus, from which emanates the septic material in so-called primary *ulcerative* endocarditis. Micrococci may set up an ulcerative process in a simple valve lesion. It is probable that a valve affected with a chronic lesion may be unable to resist the invasion of pyogenetic micro-organisms circulating in the blood. It is not necessary that such organisms should gain access to the blood immediately preceding the manifestation of the endocarditis. We believe that various forms of pyogenetic bacteria may remain latent in the body for a long time, and so that form of bacteria which is to produce the ulceration on the endocardium may have entered the body and have been stored in some tissue capable of resisting its pathogenic influence. At last some condition arises which leaves it free to gain access to the general circulation, when it attacks the diseased valves with great virulence.

This view is supported by the conclusions of Gorwitz, already given, as also by Anders, who says: "In purely septic diseases ulcerative endocarditis forms but a part of the serious general condition. Here the cardiac element serves to facilitate the generation and rapid diffusion of the poison; and since the latter is prone to attack the valve segments, the morbid lesions within the heart not rarely constitute a chief pathologic factor in septico-pyemia."

(Everything except the main points in the history of the following case has been eliminated.)

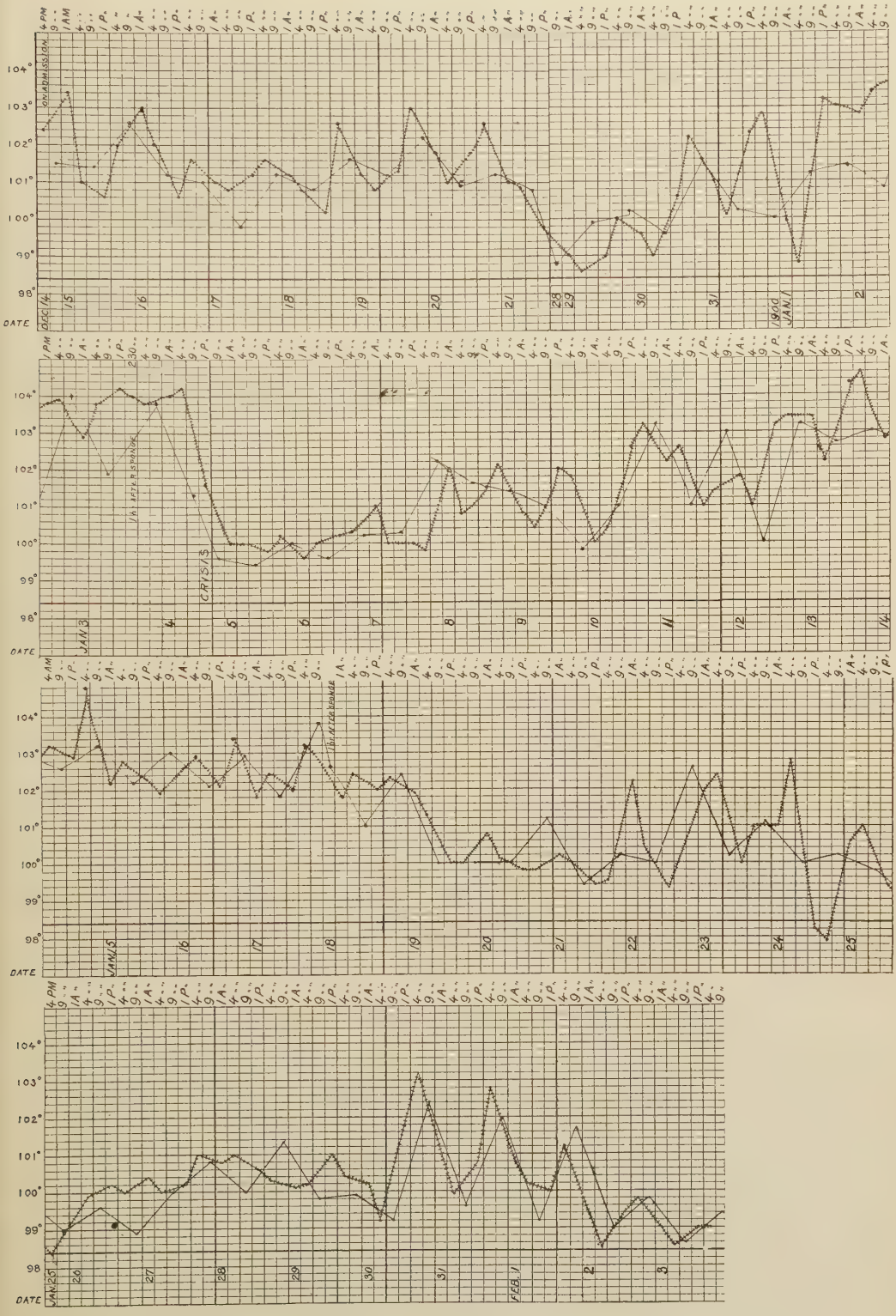
Adams. Female, six years. Admitted December 14, 1899

Family History.—Negative. Always delicate. At four years measles; good recovery. Health good until August, 1899, when treated in this hospital for remittent fever, æstivo-autumnal type; not severe, but weakening. No cardiac lesion. Discharged, cured, September.

December 1st.—Severe chill, fever, sweat, intense headache. Next day another chill. Æstivo-autumnal parasite found. Quin. sulph.

December 9th.—Tired, quiet all day.

December 15th.—Temperature 103.4°. Dyspnoea, brief unconciousness, continuous high temperature, 104°, pain in abdomen and præcordium. Well-nourished, very anæmic, distressed look. Respiration rapid, not embarrassed. Chest well formed, equal expansion. Slightly impaired percussion resonance, right scapular angle. Loud broncho-vesicular breathing, left apex; almost bronchial, right scapular angle; many râles, moist and crepitant. Breath offensive, appetite poor, tongue coated, bowels constipated. Throat, abdomen, liver, spleen,—negative. Pulse full and bounding, impulse heaving. Marked apical thrill; beat diffused over space one inch in diameter; area of dulness but slightly increased. Harsh, strong, presystolic murmur at apex, not transmitted. Second sounds much accentuated, especially pulmonic. Urine diminished, thick, cloudy; many amorphous urates; no albumin nor casts. No pain. Sleeps poorly. Interrogated: "Feels all right."



Temperature Chart of Case.

December 16th.—Temperature range 101° to 103°. Slept fairly. No urine at night. Pulse rapid and strong. Heart murmur slightly changed, double at apex. Small area dulness, left scapular angle, with bronchial breathing.

December 18th.—Temperature same, irregular, evening rises. Presystolic murmur and thrill disappeared. Soft, blowing, systolic murmur, transmitted to axilla: other valve sounds clear and distinct; pulmonic second still markedly accentuated.

December 20th.—Same condition, with slight to-and-fro friction at base; action tumultuous and plunging; apex beat much diffused; dulness much increased.

December 22d.—Examination by Dr. Wm. B. French for plasmodium malariæ, negative. Slept poorly past two nights, vomiting nourishment several times. Stopped quin. Pulse weak, rapid, at times irregular. Given tincture of digitalis. Only voids eight ounces urine in twenty-four hours.

December 26th.—Temperature normal, respiration rapid and labored. Sleeps better; does not vomit.

December 30th.—Temperature, 1 P.M., 102°; respiration as before. Pulse full and bounding; heart-sounds muffled, with marked double murmur at apex, change coincident with rise of temperature.

January 1, 1900.—Temperature intermittent, 98.8° to 103.4°. Oral mucous membrane shows small purplish spots. Respiration very labored. Pulse weak and irregular at night, full and bounding during the day. Retains all nourishment. Splashing sound at base, marked systolic murmur at apex transmitted to axilla, second pulmonic markedly accentuated; impulse heaving and diffuse. Child sleeps with eyes half closed; breathes altogether through mouth; rests well; no pain.

January 3d.—Single systolic apical murmur, transmitted to axilla. Perspires freely about head and neck, has had several drenching sweats in the past twenty-four hours. Spots disappeared from mouth. Retains nourishment. Bowels constipated.

January 5th.—Has vomited a great deal, clear green fluid, streaked with blood. Refuses nourishment. Does not sleep at night. Cries a great deal, but no pain complained of. Temperature dropped by crisis being 99.8°.

January 7th.—Nourishment retained. Urine still diminished in quantity, quality same. Face slightly swollen. Pulse full but compressible, at times weak and irregular. Sleeps much better.

January 8th.—Temperature commenced to rise.

January 9th.—101° to 102°; other conditions unchanged.

January 11th.—Temperature still elevated. Pulse and respiration rapid and full. Cried several times during night; no pain complained of. Murmur same, loud, blowing; obscures valve sounds; splashing audible at base.

January 13th.—Sleeps poorly, seems gradually weaker. Cough frequent. Temperature about 103°, with frequent excursions to 104°. Urine still diminished; perspires freely; marked odor of urine. Numerous moist râles, large and small; diminished breath-sounds at bases, rough elsewhere.

January 15th.—Yesterday, pain in cardiac region increased on coughing. Vomits nourishment frequently. Otherwise same.

January 17th.—Restless at night, crying and picking at face and bedclothes; complains bitterly of pain, stabbing in character, in region of heart; tenderness on pressure. Cough frequent and hard. Nourishment retained. Stools involuntary, large, and loose.

January 19th.—Slept better, less pain. Temperature dropped to 100°. Cough frequent. Urine diminished.

January 23d.—Stools involuntary, large, yellow, undigested. Temperature intermittent, 100° to 103°, rise in evening. Cough frequent and hard, no expectoration. Splashing not heard; sounds still obscure; churning sound; impulse forceful and heaving; apex beat much diffused. Lungs same.

January 25th.—Culture from vein in arm inoculated upon three blood serum slants. After twenty-four hours' incubation, shows numerous streptococci and another unrecognized bacillus. Dr. French.

January 26th.—4,500,000 red blood corpuscles per c.m. Hæmoglobin 62.5%. Leucocytosis. Dr. Thomas.

January 27th.—Photographed; cardiac area and apex impulse outlined with ink. Sounds changing somewhat: impulse not so heaving; churning not so audible; valve sounds more distinct; murmur not so prominent, nor does it obscure sounds so much. Temperature continues about 100° to 101°.

January 31st.—Temperature made another rise this A.M., but dropped quickly to 99.6°. No pain. No other change noted.

February 10th.—Cardiac area much increased; impulse heaving, and widely diffused, noted over whole cardiac area. First sound obscured. Murmur loudest at apex, traced to left scapular angle. Systolic splashing and churning not heard; slight rubbing at base. Sleeps well, and takes all nourishment.

February 20th.—Condition of heart unchanged. Has whooping-cough, ten to fifteen paroxysms daily which greatly exhaust her.

Case 1.—Kirkes. Male, fourteen years. Petechiæ, ecchymosis, and congestion marked and general. Auricular surface mitral valve, whitish fibrinous vegetations. Aortic, large, warty masses festooned ventricular surface; central portions, contiguous halves of cusps ulcerated and hanging loose.

Case 2.—Heineman. Male, fourteen years. Purpuric eruption general, mitral vegetations. Apex, anterior wall left ventricle, small abscess. Mitral exudation and punctate abscess of kidney, "probably contained micrococci."

Case 3.—Okazawa. Male, eight months, taken ill August 9th, with vomiting, drowsiness. (15th) Suffocative attacks, which recurred until death, 28th. Pericardium distended; heart greatly enlarged; endocardium rough, tricuspid cannot close tightly; mitral more diseased; tricuspid, disease of more recent origin.

Case 4.—Smith. Female, five and a half years. "Acute miliary tuberculosis with possibility of typhoid." Petechiæ. Vegetations, mitral, and wall left ventricle. Polypus, rod-shaped bacteria. Deeper tissues of valve nearly normal. Neither heart muscle nor liver show micrococci.

Case 5.—Harris. Male, four years. L. E. internal squint, ptosis. Pulmonary valve, extensive ulceration. Patent foramen ovale, communication between right and left ventricles. Numerous pulmonary infarcts.

Case 6.—Osler. Male, eleven years. Chorea. Left arm powerless. Right arm and leg constantly twitching. Ecchymosis. Mitral vegetations large, soft, grayish white. Spleen and kidneys, many recent infarcts. Embolic abscess, right corpus striatum (lenticular nucleus).

Case 7.—Cutler. Child, six years. Loud systolic murmur. Circulation in arm suddenly ceased. Femoral artery plugged, severe pain. (Diagnosis not made?)

Case 8.—Prudden. Female, fourteen years. Twitching muscles, left side. Brain, small hemorrhages. Tricuspid and mitral, closely adherent vegetations with loosely hanging thrombi. Left papillary muscles, small erosion. Spleen and liver, infarcts. Staphylococcus pyogenes aureus numerous.

Case 9.—Hebb. Male, eleven years. Front of pons and inferior cerebellar fissure, greenish, viscid pus. Abscesses: left frontal, corpus striatum, and optic thalamus, Lateral ventricle, walls eroded. Suppurative pleuritis, Both upper lobes, few large cavities; thick, viscid, stringy pus. Between inferior and superior venæ cavæ, right auricle, firm yellow vegetation.

Large circle : Area of relative dulness of heart. Small circle : Diffusion of apex beat. Stars : Area of absolute heart dulness.
A : Area of dulness and bronchial breathing.

Case 10. — Hebb. Male, fourteen years. Mitral and tricuspid vegetations. Upper third, right lung, recent hemorrhagic infarct.

Case 11. — Cheadle. Female, eight years. Convulsions, three attacks at intervals. Left hemiplegia. Mitral, polypoid vegetations. Ulcerations above roots of flaps. Infarcts in kidneys, spleen, and right middle cerebral artery.

Case 12. — Taylor. Female, three years. Vaginitis. Pulmonary endocarditis. Loud systolic bruit. Fluctuating temperature. Infarcts in lung.

Case 13. — Taylor. Female, eleven years. "*Remitting Fever.*" Mitral endocarditis. Systolic bruit. Optic neuritis. Streptococcus pyogenes in blood. Spleen and kidneys, infarcts.

Case 14. — Taylor. Female, twelve years. Rheumatism. Mitral murmur. Hemorrhage into brain. Spleen and kidneys, infarcts.

Case 15. — Taylor. Female, thirteen years. Chorea. Mitral regurgitant. Hemorrhage into brain. Spleen, infarct.

Case 16. — Taylor. Male, fourteen years. Mitral regurgitant. Optic neuritis. Hemiplegia, vessels obstructed. Spleen and kidneys, infarcts.

Case 17. — Ashby and Wright. Female, eleven years. Chorea and rheumatism. Murmur varying, presystolic and systolic. Complete hemiplegia, facial paralysis same side. Mitral vegetations; patches, surface left auricle and ventricle. Kidneys and spleen, infarcts. Emboli: junction right mid. and anterior cerebral, and lenticular striate arteries.

Case 18. — Pitt. Infant, eleven months. Fungating aortic endocarditis, anterior valve sloughing, two aneurysmal pouches projecting towards aorta, not perforated. Youngest case he has found, both as to aortic lesion and fungating type.

Case 19. — Gorwitz. Male, eleven years. Rheumatism. Infectious endocarditis. Recovery in two months with persistent cardiac lesion.

Case 20. — Gorwitz. Male, fourteen years. Infectious endocarditis with multiple articular suppurations. Duration nine weeks. Old cardiac lesions; slight, recent. Lesions of other organs insignificant.

Case 21. — Coulon. Female, three years. Typhoid; myoendocarditis.

Case 22. — Coulon. Female, five and one-half years. Infectious endocarditis of grippal origin.

Case 23. — Coulon. Female, nine years. Myoendocarditis in course of typhoid.

Case 24. — Fruitnight. Female, eleven years. ("Typhoid"?) Ecchymosis. Tricuspid and mitral vegetations and ulcerations. Staphylococcus pyogenes aureus almost pure, extending through fibrous tissues to heart wall. Kidney infarcts.

Case 25. — Rotch. Male, four years. Rheumatism. Chorea. Mitral vegetations and erosions. Streptococci here, in fibrin, also from lungs, pericardium, bronchial lymph glands, and spleen. Councilman traces infection from heart to pericardium, mediastinum, venous thrombi, lungs.

Case 26. — Sainsbury. Male, thirteen years. Thrill and double apical murmur. (June 22) T. 103.2° F. (24) violently delirious. T. 100–101°; no streptococci in blood. (25) Rusty sputum, no rigor. (26) Symmetrical erythema, general, five days. (July 1) Streptococci in blood. (9) General improvement. (20–24) Rash returned, cardiac dulness increased. (28) T. 104.4°; no rigor, but vomiting, cough, bloody sputum. (Aug 5) Worse; sputum blood-streaked and clot; thought spleen palpable. (17) 20 c. c. antistreptococcic serum. T. 99.8°, no local nor general reaction. (18 and 20) 10 c. c. serum; improvement; skin peeled and cleared. (22 and 25) 10 c. c. serum. (26) Rash and much itching. No streptococci. (Sept. 1) 10 c. c. serum, slight reaction local and general. T. 102°, patient seemed ill, pain at puncture, 24 hours. Recovery.

Case 27.— Wood. Female, five years. Petechiæ, convulsions, optic neuritis, retinal hemorrhage. Spleen and kidney infarcts. Numerous cerebral hemorrhages. Septic thrombus, entrance left Sylvian fissure.

Case 28.— Kanthack and Tickell. Male, three years. Septic empyema. Ulcerative tonsillitis. Otitis media. Typical, large white kidneys.

Case 29.— K. and T. Male, eight years. Pus under endocardium. Acute necrosis. Pyogenetic infection (traumatic).

Case 30.— K. and T. Female, eight years. Old cardiac disease. Secondary pneumonia. Aneurysm. Endocardial ulceration. No primary focus found,

Case 31.— K. and T. Female, eight years. Chorea.

Case 32.— K. and T. Male, nine years. Old cardiac disease. Bronchiectasis. Pericardium and pleura adherent.

Case 33.— K. and T. Female, thirteen years. Head not examined. Pyæmic infarcts. No focus found.

Case 34.— K. and T. Female, thirteen years. Otitis media. Pus in joints, spleen and middle ear. Streptococci.

Case 35.— K. and T. Female, thirteen years. Old cardiac disease. Subdural hemorrhage. Clot in internal carotid. No focus. Began with acute rheumatism.

Case 36.— K. and T. Male, fourteen years. Old cardiac disease. Amyloid spleen, infarcts. Staphylococci in blood; micrococci in vegetations. No focus found.

Case 37.— K. and T. Male, fourteen years. Caseous tubercle. Adherent pericardium. Suppurating cervical gland.

Case 38.— K. and T. Female, fourteen years. Old cardiac disease. Rheumatic history. No focus found.

Case 39.— von Leyden. Male, thirteen years. Traumatic endocarditis. Purulent pericarditis.

Case 40.— d'Astros. Male, ten months. Diphtheria. Only 10 c. c. serum given. Next day no B. Loeffleri, but almost pure staphylococcus. Soft, prominent, red mitral granulations, auricular surface, near insertion chordæ tendineæ.

Case 41.— d'Astros. Male, six years. Influenza. T. 104° F., eight days. Mitral, marked hypertrophy. Pulse 100–104, eight months after original influenza.

Case 42.— d'Astros, Male, seven years. Erysipelas. Auscultation, nothing abnormal at first. T. reached 106.8° F., pulse 142. Two injections, 10 c. c. each. Marmorek. Recovery. Persistent mitral lesion.

Case 43.— Davies. Female, eight years. Slight paresis, left lower side face. Blood twice examined, interval one month, no streptococci. Mitral ulceration and fungation— especially posterior surface auricle. Some chordæ tendineæ in aortic curtain ulcerated through. Spleen and kidneys, infarcts.

Congenital Cases.

Case 44.— Ayrolles. General endocarditis. Mitral vegetations, auricular surface; orifice obliterated. Tricuspid closed.

Case 45.— Bidone. Maternal erysipelas. Male fœtus infected per placentam. Cultures taken when cord severed with sterilized instruments. Streptococcus pyogenes. Death nineteen hours after isolation from mother.

Case 46.— Sansom. Infant died shortly after birth. Numerous vegetations on all valves, right more than left.

TABULATED STATEMENT OF PUBLISHED CASES:

DATE.	AUTHOR.	MALE.	FEMALE.	NOT GIVEN.	CASES.
1852	Kirkes	14 yrs.			1
1881	Heineman	14 yrs.			1
1884	Okazawa	8 mths.			1
1884	Smith		5½ yrs.		1
1885	Harris	4 yrs.			1
1885	Osler	11 yrs.			1
1886	Cutler			6 yrs.	1
1887	Prudden		14 yrs.		1
1888	Hebb	11 and 14 yrs.			2
1890	Cheadle		8 yrs.		1
1891	Taylor	14 yrs.	3, 11, 12 & 13 yrs.		5
1893	Ashby & Wright		11 yrs.		1
1893	Pitt			11 months	1
1894	Gorwitz	11 and 14 yrs.			2
1895	Coulon		3, 5½, and 9 yrs.		3
1896	Fruitnight		11 yrs.		1
1896	Rotch	4 yrs.			1
1896	Sainsbury	13 yrs.			1
1896	Wood		5 yrs.		1
1897	Kanthack & Tickell	3, 8, 9, 14 & 14 yrs.	8,8,13,13,13,14 yrs		11
1897	von Leyden	13 yrs.			1
1898	d'Astros	7 & 6 yrs. & 10 mths.			3
1898	Davies		8 yrs.		1
1900	Adams		8 yrs.		1
					44
1885	Ayrolles			Congenital .. 1	
1894	Bidone			Lived a few hours ... 1	
1898	Sansom				
				Congenital ... 3	3
					47

Congenital	cases	3
Males	"	21
Females	"	21
Sex not given	"	2
		47

Ages of males: 8 months, 10 months, 3, 4 (2), 6, 7, 8, 9, 11 (3), 13 (2), 14 (7) years ... 21
Ages of females: 3 (2), 5, 5½ (2), 8 (5), 9, 11 (3), 12, 13 (4), 14 (2) years 21

While the examination of the blood and the cultivation of micro-organisms in nutrient media are of prime importance, the diagnosis must still rest upon the clinical features. It is, however, of great importance to have in mind the irregular fever, the changing character of the cardiac murmur, and its tendency to form emboli; but there may be few or no symptoms referable directly to the heart, and it may simulate malarial or other septic affections. Williams says that, except in fœtal life, the left side of the heart is that usually affected, and when the right side is attacked, it is due to secondary infection.

Although the prognosis is unfavorable for recovery in most cases, nevertheless a few survive with permanent impairment of the heart valves.

The treatment must depend mainly upon food and stimulants. Sainsbury, Pearse, and d'Astros injected antistreptococcic serum with apparent benefit. Sir Douglas Powell tried in five cases the subcutaneous injection of yeast; and still another injected nuclein. The results were not such as to encourage others to follow their example.

In conclusion, I wish to acknowledge my indebtedness to Dr. Murray Galt Motter for collating the literature of the subject.

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THE VISCERAL LESIONS OF THE ERYTHEMA GROUP.

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IN December, 1895,[1] I published a series of eleven cases characterized by:

I. Polymorphous skin lesions: (*a*) acute circumscribed œdema; (*b*) urticaria; (*c*) purpura; and (*d*) ordinary exudative erythema.

II. Polymorphous visceral lesions: (*a*) local serous or hemorrhagic exudate in the walls of stomach or bowels, causing (1) crises of pain and (2) hemorrhages; (*b*) acute nephritis; and (*c*) certain rare pulmonary and other lesions.

III. Infiltration of synovial sheaths, peri-articular tissues, and arthritis.

It would have been better, as some of my dermatological friends suggested, not to have described the cases under the name erythema exudativum multiforme, the term which has been used to designate the so-called pure type of polymorphous erythema, but it was really very difficult to find a name under which to group the cases. Duhring has suggested that the majority of them should be regarded as purpura rather than erythema, but in only Cases 6, 7, and 11 was purpura the sole lesion, while in the remaining cases there was exudate (either serous or hemorrhagic or both), with swelling.

In the following communication I shall give the subsequent history of Case 2 of the first series, details of seven additional cases, an analysis of the symptoms of the whole series, and shall then discuss briefly the relations of certain members of the erythema group of skin diseases.

I will first call attention to the extraordinary series of symptoms presented by Case 2, the condition of which was reported on to the end of 1895. Briefly summarized: In his tenth year this lad had severe attacks of colic, a very barking cough, and one attack of urticaria. In

[1] *American Journal of the Medical Sciences.*

his eleventh year he had colic with an outbreak of urticaria and purpura; later a well defined localized œdema on the back of one hand, marked signs at the apex of the right lung, and an enlarged spleen. In his twelfth year he had one severe attack of colic, and the pulmonary features became more pronounced. In his thirteenth and fourteenth years he began to present the picture of emphysema, and the pulmonary symptoms were dominant. In his fifteenth year he had pronounced emphysema, dilatation of the heart, and pericarditis, of which he died. So far as the skin lesions in this case were concerned, successive physicians in the series of attacks might have diagnosed urticaria, purpura with urticaria, angio-neurotic œdema, and well defined exudative erythema.

Case 2 (of Series 1, continued).—W. E. B.[1] *Colic with urticaria in tenth year. In eleventh year attacks of colic, urticaria with purpura, angio-neurotic œdema, exudative erythema, enlarged spleen, cough with local signs at right apex. In twelfth year colic, enlarged spleen, cough. In thirteenth year colic, cough. In fourteenth year pulmonary symptoms dominant, signs of emphysema. In fifteenth year emphysema well marked, broncho-pneumonia, pericarditis, death.*

Good Family History: No rheumatism; healthy, well grown, active, and intelligent; no serious illness; fairly good digestion.

Tenth Year (1893): Attacks of severe *colic*, of such intensity that he would roll on the floor. Several attacks of epistaxis. In the summer of 1893 an attack of *urticaria*. In the latter part of the summer a very barking cough, suspected to be whooping-cough.

Eleventh Year (1894): First seen by me March 10th. Well nourished boy, a little pale; no skin lesions. The spleen was enlarged and extended in the parasternal line nearly to the level of the navel. There was moderate anæmia, 4,000,000 red blood corpuscles, hæmoglobin about 80 per cent. The urine was normal. At the right apex and in the right upper axillary region there were medium-sized moist râles; no change in the percussion note. When I first saw the case I was completely puzzled. The enlarged spleen, with a slight anæmia, raised the suspicion of some primary blood affection, but the cough, which had been an important feature, and the localized signs at the right apex, suggested, with the anæmia, the onset of tuberculosis. The sputum (which was muco-purulent) was examined carefully, but no bacilli were found. In the middle of April the cough became much aggravated. Throughout May he improved very much. In the middle of the month he had slight pains about the knees, and on the 18th there were one or two bluish stains of fading *purpura*. On May 22d, when I saw him, he was in the midst of an attack of *urticaria* and *purpura*. This, with the colic, gave me a clue to the possible nature of the case. Early in June he had an œdematous swelling, without redness, on the back of the left hand, like the ordinary lesion of *angio-neurotic œdema*. On June 6th, both ankles were swollen, and the skin of the legs presented the remains of purpura with urticaria. On the back of one hand there were patches of *erythema with exudation*. The spleen was smaller. The patient had a good summer. I saw him October 30th, and he looked very well. The râles had disappeared at the right apex.

Twelfth Year (1895): March 9th. He had been at school and very well through the winter. Last night he had a very severe attack of colic. He had a good deal of cough, and there were numerous piping râles at the apex of the right lung. The edge of the spleen could be felt. There were no skin lesions. Early in June the cough became very much

[1] The early history of this case is given fully (Case 2) in the paper referred to.

more troublesome, with severe paroxysms at night. Over the apex of the right lung in front and behind, the percussion note was of higher pitch. There were numerous moist râles over the whole infraclavicular, mammary, upper axillary, and suprascapular regions. The breath sounds were harsh, not tubular. The spleen could be felt a full hand's breadth below the costal margin. Throughout the summer he remained very well. On October 21st, the spleen was only just palpable. The cough, which had almost disappeared, had recently returned. The right apex had become much clearer: there were a few râles in the right lower axillary region, and also in the left lower mammary region. He had not been a mouth-breather and the nostrils were wide and normal.

Thirteenth Year (1896): This was marked by only one attack of severe colic in April, of great intensity, requiring morphia for its control. He went north for the summer. In October he had a recurrence of the cough, more severe perhaps than ever before. It was chiefly nocturnal, though sometimes of such severity in the morning that he would vomit his breakfast.

Fourteenth Year (1897): January 22d. He looked well and had grown. I had not seen him for nearly eight months, and I was much impressed with the change in the shape of his thorax, which had become more rounded, looked like a typical emphysematous chest, and the neck muscles stood out prominently. The resonance was impaired on and below the right clavicle. There were a great many coarse, mucous râles in front and behind at right apex, and the breath sounds were harsh, and had lost the breezy quality. Expiration everywhere was somewhat prolonged. The left lung was clear. He had had no fever. For the first time since I had seen him the edge of the spleen could not be felt. I was impressed with the condition of the lung, as the local signs at the apex seemed so pronounced. Dr. Futcher reported that the sputum, which was muco-purulent, contained no tubercle bacilli. The urine showed no change. He spent February and March in North Carolina, with great benefit. The cough disappeared entirely, and he came back looking very well. He had grown and thriven in every way. Though he was free from cough, there were very well defined signs in the lungs, numerous crackling râles at both apices, most extensive on the right side. In the right axillary region there was a friction sound. The summer was spent at Martha's Vineyard. He had no cough, gained in weight, had a good color and a good appetite. In October, November, and December the condition of emphysema progressively increased. I saw him again December 13th. There was elevation of the chest during inspiration and the neck muscles stood out prominently. Expiration was everywhere prolonged at the right apex. There were numerous bubbling râles, very similar indeed to those heard on the occasion when I first examined him. There were piping râles on expiration at the left apex. At the bases of the lungs the breath sounds were clear and breezy.

Fifteenth Year (1898): During February I attended the patient with Dr. Lockwood for two weeks in a severe attack of diffuse bronchitis with areas of broncho-pneumonia. The temperature ranged from 101° to 104.5°, and for two days he was very ill. The heart was not affected. The fever gradually subsided, and he convalesced very slowly. The attack left him with a very weak, feebly-acting heart. In April he was able to go to Pinehurst, where he remained for a month, and returned in much better condition. In May, shortly after his return, he became very short of breath. There were signs of advanced emphysema; the percussion note was very low on the right side, the heart dulness was obliterated, and there were loud, piping râles with inspiration and expiration. He passed very restless nights, and would frequently have to sit up in bed for hours at a time. During the last week in May dropsy began —swelling of the feet and legs, which gradually increased, and in the first week in June ascites. The pulse was very rapid and feeble, and there was a systolic murmur at the apex. The urine was scanty, but contained neither albumin nor tube casts. On June 16th, Dr. Lockwood detected an acute pericarditis. On the following night he was very restless, and could only breathe comfortably with the head held far forward. On the following day when I saw him there was a very loud pericardial friction over the whole cardiac region. He was much cyanosed. The pulse was extremely rapid and irregular, and he died that night.

Second Series.

Case 12.—*Neurasthenia ; dilatation of the stomach ; colic for two years at intervals ; exudative erythema ; leucocytosis.*

Berta L., aged twenty-four (Hospital No. 18,151), admitted December 19, 1896, complaining of pain and soreness in the abdomen. She had been a very nervous girl, and had had a good deal of uterine trouble. For four years she had had dilatation of the stomach, for which she had used the stomach tube. She had lost much in weight.

The patient was extremely neurotic, and impressed us as a case of very severe neurasthenia with dilated and depressed stomach. She was thin and sallow, the face pigmented. On admission she had a most extreme grade of dermatographia. The slightest scratch was followed by intense erythema and sometimes urticaria. Two days after admission, on the morning of the 22d, she had an extensive erythematous rash over the face, arms, and thighs. Upon the skin of the thighs and arms there were large raised red areas, in places diffuse, in others isolated, like big wheals. In the afternoon she had a violent attack of abdominal colic without nausea or vomiting, which lasted for about half an hour. The temperature on admission was 102°, and for four days ranged from 99° to 102°. She had no chills. There was considerable increase in the area of splenic dulness. The leucocytes were 13,450. There was no albumin in the urine ; no tube casts. On questioning the patient she says that for two years she had been liable to these attacks of colic, which came on with great suddenness. This was the first one in which it had been associated with any skin lesions. The erythema persisted for about two days and then disappeared. It did not extend to the face or hands.

Case 13.—*Attacks of colic every week or ten days for six months ; on admission typical lesions of erythema exudativum multiforme ; high fever ; improvement ; recurrence ; pains in the joints ; arthritis in one joint of finger.*

Ruberta F. B., aged forty-nine (Hospital No. 19,713), admitted June 14, 1897, complaining of pains in the hands and a skin rash. There was no rheumatism in the family.

Personal History : She had had the ordinary diseases of childhood and once urticaria. No rheumatism. When twenty-six years old she had severe diarrhœa with bloody stools ; was ill five weeks. She has had six children. In November, 1896, she was operated upon successfully for an ovarian tumor.

Present Illness : One week after the operation she had an attack of severe abdominal pain, limited to the epigastrium, which came on very suddenly. While in hospital she had three attacks, all requiring morphia. Since dismissal she has had an attack every week or ten days. Twice she has had two attacks in a week. She has had no vomiting, but she is always slightly nauseated. The intensity of the pain is such that she cries out at the top of her voice. She always has fever with the attacks. The pain comes on, she says, "as suddenly as one could blow out a light," and leaves her just as quickly.

On June 3d, she had a slight chill, and has not been well since. On the morning of June 12th, she noticed a few small red papules on the left hand. About 2 P.M., she had a slight chill, followed by fever, with sudden pains about the joints, and an eruption appeared in many regions of the body. Her eyes became red and painful. On the 13th, the eruption became more raised and confluent. She had considerable pain at the onset, no vomiting, but some nausea, no diarrhœa.

On admission she was a well nourished, stout woman ; conjunctivæ deeply injected ; temperature, 104.5° ; tongue coated. The skin presented most extensive lesions of erythema multiforme. Face and neck presented many raised, red, infiltrated areas, some of which were surmounted by small vesicles. On the legs and thighs there were deep red blotches, the smallest about 1 cm. in diameter, the largest about 2 cm., raised and in places looking like erythema nodosum. On the hands and forearms the rash was infiltrated, the spots less red than those on the legs and face, and surmounted by large, clear vesicles. On the hands there were large, confluent, indurated, deep red areas, which were capped in places with hemorrhagic vesicles. The first phalanx of the right forefinger was swollen, deeply cyanotic, and

surmounted by many vesicles. The skin of the thumb of the right hand presented a few hemorrhages. The fingers were excessively tender. It was noted as an interesting point that with the extensive lesions on the hands there was no tenderness or swelling of the epitrochlear glands or of those in the axillæ.

During the first two days the temperature ranged from 102° to 103°. The rash persisted; many of the infiltrated areas on the hands and arms became capped with small blebs, the contents of which in places became purulent. The hands and fingers were greatly swollen. The legs showed fresh outbreaks of erythema; no vesicles. The edge of the spleen was palpable. There was no involvement of the heart. The temperature fell on the 19th. Fresh crops appeared on the 17th and 18th, and on the former date she complained of much soreness in the joints. Unfortunately the urine chart was mislaid.

Case 14.—*Man aged fifty-seven; from twentieth year every few months attacks of nausea, vomiting, and abdominal pain associated with outbreaks of urticaria; no hemorrhages from the mucous membranes; final attack with purpura and urticaria; much vomiting and profuse and fatal hemorrhage from the stomach, with blood in the urine and the passage of blood from the bowels.*

This very remarkable case in a physician, aged fifty-seven, I saw on May 31, 1898, with Dr. Wilkins. He had been a temperate man, of good family history. He had worked hard, and had been singularly free from diseases, except the one to be described. He had never had rheumatic fever.

From his twentieth year he had had attacks of urticaria of the greatest intensity. As a young man they occurred at long intervals, but during the past five years he has rarely passed three months without an attack. Each one was associated with nausea, vomiting, and great pain and tenderness in the epigastric region. The eruption usually appeared in broad patches, and covered almost the entire surface of the body. It persisted from the outset to the close of the attack, which lasted usually from a week to ten days, or even two weeks. After the outbreak the vomiting and pain would subside, but the urticaria would persist. Subcutaneous or mucous hemorrhages did not occur until the last attack. No microscopical examination of the urine was made until the summer of 1897. For the past two or three years the attacks have been associated with great depression of the circulation. The temperature rarely rose above normal. In July, 1897, Dr. Wilkins found albumin and tube casts in the urine, and detected marked sclerosis of the peripheral arteries.

Dr. Wilkins, who attended him for years, said that it was impossible to appreciate the intensity of the attacks without seeing them. The nausea, vomiting, and abdominal distress, the itching of the skin, and the persistence of the attacks for a week or ten days, made his life wretched; yet he bore up bravely and attended to a large practice.

The final attack began April 25th, and followed the usual course until May 1st, when it was complicated with a bronchitis. On May 20th he went to Atlantic City, and while there, May 26th, for the first time he vomited blood. He returned home May 30th, and at 1 A.M. on the 31st he had a profuse hemorrhage from the stomach.

I saw him on the morning of the 31st with Dr. Wilkins. He was well nourished, but looked pale and sallow. On the left ear there was a small tophus. The tongue was clean, but presented on the right margin two spots of hemorrhage, one of which had broken and was oozing blood. Beneath the skin of the arms, trunk, and legs were many bluish-black subcutaneous hemorrhages. On the back there were at least a dozen, ranging from 2 to 5 or 6 mm. in diameter. On the outer surface of the right thigh there was a localized solid infiltration of both skin and subcutaneous tissues, forming a hemorrhagic nodule the size of a walnut. On the radial side of the left arm there was a very extensive fading ecchymosis. On different parts of the body there were small petechiæ. The apex beat was outside the nipple line. There were marked hæmic murmurs. The urine showed a moderate amount of albumin and a few tube casts. The radials were sclerotic; pulse a little rapid.

On Wednesday, June 1st, he became very much worse. He had epistaxis; the purpuric

spots increased; he had vomiting of blood, hemorrhage from the bowels, and blood appeared in the urine. He became very feeble, and died early on the morning of June 2d. There was no autopsy.

Case 15.—*When a lad, one attack of hemiplegia with aphasia lasting for a week; within a year five or six attacks of transient hemiplegia; history of migraine in 1896, and a mild attack of rheumatism; angio-neurotic œdema of the upper lip; outbreaks of urticaria; in 1897, attack of abdominal colic, with pains in the legs and an outbreak of purpura and urticaria; in 1898, hæmaturia and albuminuria.*

C. A. R., physician, aged twenty-nine, a large, robust man, weighing above two hundred pounds, consulted me Feb. 10, 1898, and gave a long history of himself, of which I give an abstract.

From childhood, indigestion. When twelve or thirteen years old, after a hearty breakfast, he had right hemiplegia and aphasia, which lasted for a week or ten days. Within the year he had five or six attacks of hemiplegia, each successive one less severe, and not accompanied by aphasia. From that time until the present, he occasionally feels a numb, tingling sensation in the side. In 1893 he had lumbago of a severe type. In 1895 a severe attack of grippe. At this time he began to suffer badly with attacks of migraine, which he has had at intervals for some years. In 1896 he had a mild attack of rheumatism, confined to bed only two or three days. In February, 1897, while in New Orleans, he had a sudden attack of swelling and pain in the feet. About the same time he began to suffer with soreness at the ends of the fingers. The upper lip would frequently swell, and he would have outbreaks of urticaria, associated with darting pains in the legs of very great intensity. In May, 1897, after exposure to a draught when heated, he had an attack of great soreness in the right iliac fossa, supposed to be appendicitis, temperature 102°, with some colic. While convalescent from this he had another remarkable attack of pain in the calves of the legs. They became so sore and tender that he could only walk with a cane, and was afraid that abscesses were beginning to form. Associated with it, however, there were large wheals of extravasated red blood. On May 26th he had another attack of nettle rash.

When I saw this patient in February, 1898, his general condition was good, he had no arthritis, and there were no signs of appendicitis.

In May, 1898, the patient had a chill, followed by hæmaturia and albuminuria, which had disappeared by June 1st. With this there was a return of the pain in the right side.

The doctor writes, "I am convinced that this attack of nephritis is only a part of my old trouble."

Case 16.—*For three months, attacks of pain in the abdomen, with vomiting, swelling of the joints; purpura; recovery.*

Harry L., aged eleven, applied at the Out-patient Medical Department March 23, 1899, complaining of indigestion, pain in the stomach, and vomiting.

Family History: Father and mother living and well; one sister living who has indigestion; one brother died of dysentery. No members of family have suffered with rheumatism.

Personal History: He was delicate as a baby. Has had measles, chicken-pox, and whooping cough. When nine years old he had some stomach trouble of doubtful nature.

Present Illness: In January, just about two months ago, the patient was taken ill suddenly with pain in the abdomen and vomiting; the attack was attributed to the food which he had eaten the night before. The symptoms persisted for nearly a week, and the patient could only retain diluted milk. The pain came in paroxysms of great severity. From this time the patient has been ill off and on, frequently having had to remain in bed with the attacks of pain, and particularly if he took any extra food. He has been weak and nervous, and has lost in weight. During the first attack in January an eruption was noticed on the legs, and at this time one ankle was considerably swollen, and later one knee.

When seen March 23d, he was just recovering from an attack. He looked pale and weak, and a little anæmic. There was nothing of note in the examination of the chest.

Neither the spleen nor the liver was enlarged. The abdomen was not swollen, nowhere tender on pressure. On both thighs and legs there were recent purpuric spots, varying in size from a pin-head to a split pea. They were not raised. The left ankle still looked a little swollen. The right knee was slightly flexed and still a little stiff. The urine showed no trace of blood or albumin.

The blood coagulation time was three minutes. He was ordered Fowler's solution, and the mother was given very specific instructions with reference to the diet of the patient.

April 10th: The patient has improved very rapidly. He has had no gastro-intestinal symptoms, the joints are now quite well, and the purpuric spots have disappeared completely.

Case 17.—*Following influenza, in January, attacks of arthritis with cramps in the abdomen and an outbreak of urticaria; eight attacks between January and May; during stay in hospital, swelling of wrists and back of hand; erythema; urticaria, spontaneous and factitious; no purpura; recovery.*

Geo. K., aged eighteen, admitted to Ward F, May 17, 1899, complaining of pains in the joints, shortness of breath, and pains in the abdomen.

Family History: Father died of an accident. Mother is living and well. There are two sisters and one brother. No similar troubles in the family. No rheumatism.

Personal History: Measles and mumps when seven years old. Never ill again until last summer, when he had chills and fever, but, on inquiry, there is no history of any definite chills. In December, he had an abscess on the back of the hand. In January of this year he was ill with influenza. He has worked hard for a year making confectionery. He does not use alcohol or tobacco. His general health has been excellent; he has never had rheumatism or chorea.

He has always bled easily from the nose, and last year, during the very hot weather he bled almost every other day.

Present Illness: While in bed in January with what was called influenza the patient had swelling and pain in the feet and hands. He has not been able to work since the middle of January, except for two weeks. During the first week in January he noticed on his legs and on the arms "lumps" like those following the bites of insects, about the size of a quarter of a dollar (2.5 cm.) in diameter. They burned and itched, and gradually disappeared. As many as a hundred came out in a crop together. With this there was some swelling of the arms and legs. At the same time he had attacks of cramps in the abdomen. He has had in all about eight of these attacks since January. They were usually very severe, causing him to cry out, and he was doubled up in them. He has noticed that he has been short of breath for nearly a month. He has had no fever.

Condition on Admission: The patient was a well nourished, healthy-looking young man. The tongue was clean; pulse regular, 80, of good volume.

The arms looked natural; no pain or swelling about the shoulders or elbows. The right wrist was somewhat swollen and painful. Both hands were swollen; looked a little cyanotic, cold, and were slightly tender on pressure; there were no urticarial wheals. No swelling over the metacarpal or phalangeal joints. There was slight tenderness of the right knee; no swelling. There was no purpura.

Heart: The maximum impulse was in the fifth interspace, outside the nipple line. There was no thrill. At the apex there was a loud, rough, blowing murmur, propagated into axilla, and heard distinctly over the whole back of the chest. At the aortic area there was a rough systolic murmur of less intensity than at the apex. The spleen was not palpable. There was no fever. The specific gravity of the urine was 1.017; no albumin, no tube casts.

May 18th: On the back of both arms I noted a slight erythema with a few wheals of urticaria. They were present also on the outer side of the left thigh. Over the back of the left hand there was a swelling without special redness. The patient had improved very much.

On May 23d he complained of pain in the first phalangeal joint of the left ring finger, which was reddened and swollen, not tender. There were two urticarial wheals on the right forearm, and one on the left. Factitious urticaria was readily produced. The blood coagulation time was four minutes fifty seconds.

May 26th : The patient had a severe attack of colic to-day at 12.30. It passed off in a few hours. There was no rise in temperature. The abdominal pain persisted at intervals for a few days.

On June 4th, the dermatographia was not so marked. There were still some urticarial blotches.

June 9th : The patient had been up and about. The joint pains had subsided. The urticaria still came out at times about the joints.

June 19th : Condition very good. Patient discharged. During his illness this patient had no purpura.

Case 18.—*During first year swelling of knees ; from second to seventh years frequently recurring attacks of pain in the abdomen with vomiting and with swelling of the knees, but no skin rash ; following vaccination, attack of great severity with extensive lesions of erythema, purpura, and urticaria ; melena ; recovery ; recurrence of the skin lesions ; enlargement of the spleen.*

Barbara P., aged seven, admitted to Ward G, March 28, 1899, complaining of pain in the abdomen and high fever.

Family History : The parents are living and well. There are three brothers and three sisters. No history of rheumatism ; no similar cases have occurred. It seems in all respects to be an exceptionally healthy family.

Personal History : The child has had measles, mumps, chicken-pox, and whooping cough. When three months old she had swelling of the knees, which the doctor called acute inflammatory rheumatism, and which lasted on and off for five months. No other joints were affected. The condition has recurred occasionally. She never had any breaking out upon the body ; never had bleeding from any mucous membranes. Since her second year she has had at intervals of a week or two gastro-intestinal attacks, characterized by vomiting and severe cramp-like pain about the navel, which would cause her to cry out. The attacks lasted for one or two days. At times during these attacks the legs would become swollen to the ankles, and the knees were tender, the left always worse than the right. They were never reddened. The mother, who seems an intelligent woman, was questioned very carefully as to these attacks, and gave always the same account. She was positive that not a month had passed since the child's second year without an attack of this cramp colic and vomiting. She has never noticed any blotches or redness of the skin.

Present Attack : Three weeks ago the patient was vaccinated, and two days later was unable to go to school on account of loss of appetite and pains in the limbs, which lasted for two or three days, and have recurred at intervals. She had no cramps, no vomiting. The present attack dates from four days ago, when she began to have fever and vomited a good deal, chiefly the food she had eaten. With this there was much pain in the abdomen. The left leg became swollen from the knee down, and a dark eruption appeared upon the skin of the legs in the form of dark brown spots about the size of a quarter of a dollar (2.5 cm.) in diameter, raised, and capped with small blisters. The spots were painful. The next day the eruption had faded, but other red spots came out about the knees, and yesterday they appeared on the back and elbows. The pain has persisted during the past four days, and she has vomited at intervals. The mother says it is the same sort of painful attacks that she has had so frequently.

Present Condition : Dr. Futcher dictated the following note : The child is fairly well nourished. Lips and mucous membranes are of a good color. The teeth are discolored, but not decayed. The tongue is coated with a slight brownish fur. Pupils equal and of normal size, react to light and on accommodation. Pulse : 108, of fairly good volume and tension,

regular in force and rhythm. Vessel wall not felt. Temperature on last admission at 10.30 last night was 99.5 °, since when it has not been higher.

On left upper arm is a very large scab, the result of vaccination sixteen days ago. The skin is slightly reddened and infiltrated about the crust. The glands in the axilla slightly enlarged. Over the right elbow are a number of slightly elevated papules which are somewhat hemorrhagic in character. The redness does not disappear on pressure. There is one small hæmorrhagic papule on the left elbow-joint. Over the right knee-joint are a number of purpuric spots. Some of these are about $\frac{3}{4}$ of a cm. in diameter. A few pin-head purpuric spots over left knee-joint. There are no urticarial wheals on the body. Vaso-motor skin reflex active, but no factitious urticaria. None of the joints are swollen, nor are any of them painful this morning; no stiffness of the cervical muscles. Post-cervical glands slightly enlarged. Left epitrochlear gland distinctly enlarged and easily palpable. Inguinals very slightly enlarged.

Thorax: Well formed and symmetrical. Lungs clear over fronts and backs on auscultation.

Heart: Point of maximum impulse visible and palpable in fourth interspace, 4 cm. from mid-sternal line. Impulse forcible; no thrill. Cardiac dulness not increased. Auscultation—first sound very loud and booming at apex; both are clear at aortic and pulmonary areas, and of normal relative intensity. No murmur in vessels of neck.

Liver: Is not enlarged.

Abdomen: Looks natural, symmetrical. Respiratory movements present. General abdominal tenderness. The pain is most severe in the umbilical region; no localizing symptoms in region of appendix. Patient says it is a little more tender in the right than left iliac fossa. There is no muscular spasm. Spleen not palpable.

March 30th: Her temperature is normal. There is a diffuse erythematous rash over the right cheek and the lower jaw, and a few discrete erythematous raised patches on the neck. The redness does not entirely disappear on pressure. On the upper and lower lips are small patches of a cherry-red color, which partially disappear on pressure. No pain or swelling of the joints. Blood cultures made by the usual method negative. The urine has a specific gravity of 1.027, no albumin, no tube casts.

March 31st: There are a few new purpuric spots over the left knee-joint. Those on the right knee and right elbow are gradually clearing. She still complains of pain in the region of the navel. On examination there is a little blood in the stools to-day.

April 1st: Tongue still very heavily coated. Over the left elbow there is a fading crop of purpura. On the right elbow there is a patch of erythematous infiltrated nodules of a deep red color, capped with dried vesicles. On the right knee I noted that there were several infiltrated patches looking like purpura urticans.

April 2d: Patient cries if the abdomen is touched. There are a few fresh raised purpuric spots on both elbows.

April 3d: Fresh purpuric spots in both gluteal regions, many of them slightly elevated.

April 4th: Fresh spots on knees, elbows, and buttocks. Yesterday the child complained much of pain. She still vomited occasionally after taking food. A few flecks of blood in the stools.

April 5th: Fine small purpuric spots appeared since yesterday on the left elbow. No abdominal pain to-day. The heart sounds are quite clear.

April 6th: Yesterday the child began to pass more blood in the stools. During the night she had several evacuations of bloody mucus and of a thin bloody fluid. She had slight pain in the abdomen. She has had about a dozen small stools this morning, all with blood and mucus.

At the bend of the left elbow there appeared since yesterday subcutaneous hemorrhages from .5 cm. to 2.5 x 1.5 cm. in diameter. They are tender to the touch and resemble recent

bruises. A number of smaller patches are scattered along the extensor surfaces of the forearm. Fresh purpura about the left elbow.

April 7th : Patient does not look so well this morning. Complains of severe pain in the abdomen. She looks a little anæmic from the intestinal bleedings, which continued throughout yesterday and last night, but are less frequent to-day.

April 9th : She had seven stools in the past twenty-four hours, fluid, greenish-black, with a small amount of thin fluid blood. Very little abdominal pain.

April 10th : Fresh hemorrhages on both surfaces of the forearms, a few on the right hand. Bruise-like ecchymoses on the palm of the right hand, and a large one on the left.

From the 12th the patient improved. There were a few fresh purpuric spots on the 14th and on the 16th, on which date too there were noticed several small ulcers over the tongue and inner surface of the right cheek.

April 22d : The patient has been doing remarkably well. On the 29th she was discharged.

Blood : A very careful study of the blood in this case was made by Dr. Gwyn, of which the following is a summary.

Red Blood Corpuscles : On admission they were nearly 6,000,000 per c.mm. After the hemorrhages from the bowels they were reduced, but did not fall below about 4,800,000.

Hæmoglobin : Eighty per cent. on admission, falling after the hemorrhages, and was only 60 per cent. on April 24th.

Leucocytes : There was pronounced leucocytis from the onset : on March 29th, 18,800 ; on March 31st, 37,000 ; on April 2d, 45,000. Then they gradually fell and were normal on April 27th. The differential count at the height of the leucocytosis (500) gave polynuclears 86 per cent., small mononuclears 6.2, large mononuclears 2.6, transitionals 2.8, eosinophiles .4, nucleated red blood corpuscles 1.

Coagulation Time, April 7th, from 1 minute 30 seconds to 1 minute 55 seconds ; April 23d, 2 minutes to 2 minutes 15 seconds.

Blood Platelets, April 13th, 122,000 per c.mm. ; April 23d, 230,000 per c.mm.

Blood Cultures, made twice, were negative.

The temperature range was between 99° and 100.5° ; even when she was quite convalescent her temperature was between 99° and 100°.

May 8th : The patient returned to-day complaining of a breaking out of spots on the legs, which came the day after her discharge. She looked a little pale, and she had numerous purpuric spots over the ankles, legs, knees, and lower thighs. Over each elbow on the olecranon there was an erythematous spot about 2 cm. in diameter, pale red, raised, with a tendency to clear in the centre. An identical spot almost completely surrounded the tip of the little finger of the right hand. The edge of the spleen was distinctly felt. There were no urticarial wheals, and no abdominal tenderness.

ANALYSIS OF THE SYMPTOMS.

I.—THE VISCERAL MANIFESTATIONS.

(a) *The Gastro-Intestinal Crises.*—This special feature in the entire group of cases presents the most distressing, though not the most dangerous, of the visceral complications. The attacks may be characterized by colic alone, more frequently colic and vomiting, colic with vomiting and diarrhœa, and lastly colic with vomiting of blood, or the passage of blood in the stools. As a rule, with the gastro-intestinal crises there are cutaneous manifestations, but not invariably. In

Case 2 there were severe attacks of the most agonizing colic without any other symptoms. Cases 1 and 18 are particularly interesting as illustrating the nature of certain obscure forms of gastro-intestinal colic, particularly in children. Case 1 consulted me for remarkable attacks, which recurred every two months, lasting for from six to ten hours, accompanied with fever and remarkable delirium. These had recurred for nearly eight years. For the first six years, with the attack he had an outbreak of what he called big liver-spots. For two years there had been no skin complications. In Case 18, for five years the child had been plagued with attacks of colic and vomiting, for which the mother had consulted many physicians. When she came under my observation the nature of the trouble was made evident by the concurrent outbreak of an exudative erythema. These crises are identical with those which occur in the angio-neurotic œdema. Several members of the family with this disease which I have described [1] had had urticaria, and the patient who came under my care had coincidentally with the angio-neurotic œdema characteristic urticarial wheals on the chest and thighs.

Case 14 illustrates the remarkable relationship which exists between urticaria and the gastro-intestinal crises. The association of digestive disturbance and hives is common enough, but these cases are, I think, somewhat different, and it is reasonable to suppose that the lesions causing the pain in the abdomen are associated with the formation of wheals and swelling in the mucous membrane of the stomach and intestines. F. A. Packard [2] has recently considered the question of urticaria of the mucous membranes in an exhaustive paper, in which many references are given to the formation of wheals in the throat and mouth. Though writers speak of involvement of the stomach and intestines, I know of no instance in which the lesions have been actually seen in these parts. Colcott Fox, in his article on "Urticaria," in *Allbutt's System*, states that wheals have been seen in the stomach of a rabbit and dog and cat. In Case 14 the patient had for years recurring attacks of severe pain in the stomach, with coincident urticaria, the skin lesions lasting for a much longer period than the abdominal symptoms. Packard refers to a case of Lemonnier, in which there had been giant urticaria and an attack of vomiting of blood, which was attributed to urticaria of the stomach. Both Pringle [3] and Chittenden [4] have reported cases of recurring attacks of hæmatemesis with urticaria.

[1] *American Journal of the Medical Sciences*, April, 1888.
[2] *Archives of Pediatrics*, October, 1899.
[3] *Clinical Transactions*, vol. xviii.
[4] *British Journal of Dermatology*, 1898.

It is interesting to note that in Case 14 the patient had hemorrhage from the stomach as a terminal symptom. In the series of eighteen cases, urticaria was present at some time or another in eight cases, not including the three cases in which acute circumscribed œdema was present.

(b) *Hæmaturia and Nephritis.*—Acute nephritis occurred in Cases 3, 4, 5, 6, 8, and 15. Chronic nephritis occurred in Case 14, and hæmaturia at the close. In a case referred to in my previous paper, reported by Dr. Prentiss, of Washington, a chronic nephritis of several years' duration followed an attack of arthritis, with purpura and gastro-intestinal crises.

To two of the instances of nephritis I may call particular attention, as death directly occurred from this complication. In Case 3, a boy of six years, the onset was with pains in the ankles, colic, and urticaria. The colic recurred with great severity. It was not until the fifth week of his illness that the urine became scanty and albuminous, and showed red blood corpuscles and many tube casts. He died with dropsical symptoms in three months. In Case 8 the disease set in with pain in one ankle and urticarial rash. Within a month the child had anasarca, with albuminuria and tube casts in the urine. The patient died in uræmic coma.

(c) *Hemorrhages from the Mucous Surfaces.*—These occurred in six cases; from the bowels in Cases 3, 6, 11, 14, and 18; from the nose and gums in Case 10; from the stomach in Cases 11 and 14; from the kidneys in Cases 11 (not associated with nephritis) and 14. Several of the cases of acute nephritis had a few red blood corpuscles in the urine.

(d) *Cerebral Symptoms.*—In two cases in the series there were remarkable symptoms pointing to involvement of the brain. In Case 1, a man aged twenty-seven, who during six years had recurring attacks of gastro-intestinal crises, with the onset of the symptoms had fever and became delirious and talked nonsense. In Case 15, it seems highly probable that the recurring attacks of hemiplegia, five or six within a year, were associated with changes in the brain of essentially the same nature as those which subsequently occurred on the lip and in skin. They remind one somewhat of the attacks of recurring aphasia with paralysis in cases of Reynaud's disease.

(e) *Pulmonary Complications.*—Only one case of the series (2) presented marked pulmonary symptoms, to which I have referred in the supplementary history of the case.

In erythema nodosum and in urticaria, asthmatic attacks have been described, due, it is thought, to changes in the mucous membranes of the bronchi of a nature analogous to those in the skin.

Packard, in the paper already referred to, reports cases of the coincidence of asthmatic attacks with urticaria, and gives a very full consideration of the literature. The two conditions have occurred coincidently, or an outbreak of urticaria may replace an asthmatic attack. In Case 2, without positive asthmatic attacks, there were constant signs of bronchial trouble, but it was not until the emphysema was well established that there were bouts of nocturnal dyspnœa.

It is interesting to note that in not one of the series of eighteen cases was there acute endocarditis, a not very infrequent lesion in certain forms of polymorphous erythema.

II.—SKIN LESIONS.

An analysis of the lesions of the skin in this series is of interest. In four (Cases 6, 8, 11, and 16), purpura alone was noted. In the remaining fourteen cases, the lesions were characterized by erythema with exudation, either urticaria or urticaria with purpura, acute circumscribed œdema, or the lesions of a typical erythema multiforme. Acute circumscribed œdema occurred in Cases 2, 7, and 15, all in association with other exudative lesions. It is interesting to note that the skin lesions may be absent for a protracted period, as in Case 1 for two years with recurring crises of great severity, or in Case 8, severe attacks for five years before any skin lesions appeared.

One of the most interesting features in these cases is the inconstancy of the character of the skin lesions; thus in Case 2 the lad had urticaria in his first outbreak, subsequently urticaria and purpura, and later an area of angio-neurotic œdema of the most characteristic form, and still later, on one hand, very typical lesions of an exudative erythema.

Case 14 illustrates an extraordinary recurrence of urticarial attacks for many years. No cutaneous hemorrhages appeared until his final and fatal illness, in which there were hemorrhages into the skin and from the mucous surfaces. In some cases urticaria has existed with the purpura, and in some, simple purpura in one place and purpura urticans in another. I have not seen the coexistence of urticaria with angio-neurotic œdema, which has been referred to recently by Oppenheimer (*Lancet*, 1898, 2), but in one case of the hereditary form which I described some years ago, urticaria had preceded the outbreaks of œdema.

This great variability in the character of the skin lesions is of considerable moment, and it is quite possible that within a year in an individual case the diagnosis might be given of simple purpura, peliosis

rheumatica, angio-neurotic œdema, exudative erythema, and simple urticaria.

III.—ARTHRITIS.

Swelling of the joints or of the synovial sheaths or peri-articular tissues occurred in ten of the cases in the series. The joint trouble may be transient, and, as in Case 2, may occur but once in a prolonged illness. There may be a polyarthritis of great intensity simulating acute rheumatic fever, as occurred in Case 4. Infiltration of the peri-articular tissues and of the subcutaneous structures over the joints may simulate a genuine arthritis. In some of the cases the swelling was chiefly along the tendons, as on the back of the hands. In one case (5) there were swelling and pain in the left biceps muscle. In other instances, as in Cases 7 and 18, there may be swelling of the foot and ankle joint and of the leg, due to extensive infiltration, œdematous or hemorrhagic. In Case 18, the attacks of swelling of the knees and legs, without any skin rash and in connection with the recurring cramp-colic, were due, in all probability, to infiltration about the joints. I have not seen subcutaneous fibroid nodules in any of the cases.

And lastly a few remarks on *the mutual relations of the members of the erythema group.*

That there is a close affinity between exudative erythema, Henoch's purpura, peliosis rheumatica, urticaria, and angio-neurotic œdema, is shown by first, the similarity of conditions under which they occur; secondly, the identity of the visceral manifestations; thirdly, the substitution of these affections for each other in one and the same patient at different times. The student is, however, at the outset confronted by this interesting feature. On the one hand, similarity of lesions may result from a variety of causes. The purpuric rash of iodism, of endocarditis, of scurvy, and of smallpox are identical. The wheals of urticaria from nettles, of an acute gastric toxæmia, and from the poison of the malarial parasites are indistinguishable. A typical acute exudative erythema may result from several causes. On the other hand, unity of cause may be associated with a variety of lesions. In one and the same person within a few months, presumably under the same etiological conditions, there may be a multiplicity of skin lesions; and, as in several of the cases here described, four or five separate diagnoses would be required to cover the cutaneous lesions at different periods. We cannot say why in one case there is exudate of red blood corpuscles without erythema (purpura), in another a serous exudate with hyperæmia (urticaria), in a third serous exudate with hyperæmia and hemorrhage in varying degrees (erythema exudativum

multiforme), in a fourth serous exudate alone (localized œdema). Two or three of these lesions may coexist or may rapidly succeed each other during the same attack, or in succeeding attacks the skin lesions may vary, urticaria in one, purpura in another, and so on.

Etiologically the cases here reported are very difficult to group. Cases 1 and 15 are peculiarly obscure, and suggest a relationship with migraine. In Case 12, associated with a dilated stomach, the cause could be looked for in all probability in the products of gastric fermentation. Case 14 belongs to that remarkable group, comprising many cases of localized œdema (as well as ordinary urticaria), in which we must suppose that during long periods of years, in association with a special vulnerability, inherited or acquired, of the vaso-motor system, there is manufactured some poison, either endogenous or exogenous (gastro-intestinal). Case 5 followed an acute infection (gonorrhœa), and comes in the category of the acute infectious erythemas, of which two forms may be recognized, a specific independent malady, which has been met with in epidemic form, and a variety which may occur in any of the acute infectious diseases. It is quite possible that some of the cases were of rheumatic origin, though I hold with those who do not look upon the arthritis, so commonly present, as necessarily indicating the presence of a rheumatic poison.

A point of special interest is the relationship of certain forms of purpura to the erythemas. Schönlein's peliosis rheumatica may be regarded as a hemorrhagic type of an exudative erythema, and Henoch's purpura, which is characterized specially by the occurrence of gastro-intestinal crises, belongs to the same group.

Several interesting communications have of late dealt with this question. The case reported by Dr. J. Fayrer[1] illustrates the sequence of arthritis, œdema, erythema exudativum purpuricum, and finally sloughing of the affected areas. The illustration suggests peliosis rheumatica. In a very important paper "On the relationship of Purpura Rheumatica to Erythema Exudativum Multiforme,"[2] after describing several interesting cases of purpura rheumatica and giving an analysis of those under his care, forty-two in number, Mackenzie says: "I now come to the *nosological position* of purpura rheumatica. The prevailing view is that purpura rheumatica is a variety of erythema multiforme, and Schönlein's original description of the disease certainly is in accordance with this conclusion. With this view I do not altogether agree. I admit its very close alliance, and that the various forms of erythema are almost as closely associated with rheumatism as is the form of purpura, to which I should restrict the term purpura

[1] *British Journal of Dermatology*, vol. viii. [2] *Ibid.*

rheumatica. But in E. multiforme the hemorrhage is only incidental, whilst in purpura it is primary and essential, and in the majority there is no co-existing erythema. Still in a small minority we find some form of erythema co-existing. In erythema nodosum we see precisely the same thing. In the majority of cases we find the eruption limited to the nodose form, though more rarely we have other forms of erythema associated with it. Thus, admitting that E. nodosum is a variety of polymorphic erythema, we recognize it as a distinct type. Some would go so far as to call it a distinct disease, but in this I do not concur. Similarly I claim P. rheumatica, such as I have sketched, as a distinct clinical type which deserves recognition, and I have endeavored to indicate the class of cases to which I think the term should be restricted." He very correctly concludes that it is undesirable to consider all cases of purpura to be essentially of the nature of polymorphic erythema.

Certain French writers, as Thibierge, in the recently issued second edition of the *Traité de Médecine* (vol. iii.), group under the erythemas both the purpuras and the urticarias, describing only three principal types of the former—the rheumatic and infectious purpura, and the purpura hæmorrhagica of Werlhof.

The relation of the so-called angio-neurotic œdema to the other members of the erythema group is, I think, less doubtful. That it is essentially of the same nature as urticaria and should be grouped with it, is shown by their simultaneous outbreak, by their substitution one for the other, by the identity of the visceral complications, and by the peculiar local limitation occasionally seen in both, as to the face or hands. I do not know if the simple urticaria has ever been described in members of the same family through a series of generations, but, as I have already mentioned, a patient under my care with the hereditary form also had urticaria. A very interesting paper on this affection has recently been published by Schliesinger, under the name of "Hydrops hypostrohus" (*Munchener med. Woch.*, Aug. 29, 1899). He groups together the various manifestations of angio-neurotic œdema,—the acute recurring œdema of the eyelids or of the lips, the acute recurring exophthalmos, certain forms of nervous coryza, the hydrops articulorum intermittens, the acute œdema of the tongue, of the pharynx, and of the larynx, certain forms of nervous asthma, the acute œdematous swelling in the stomach and the intestines (causing recurring attacks of colic and the intermittent vomiting of Leyden), intermittent forms of nervous diarrhœa, and, lastly, certain affections of the kidneys causing polyuria or hæmoglobinuria. There is really no warrant for separating too sharply angio-neurotic œdema and urticaria; Oppenheimer

has seen them occur together, and many observers have noticed the interchange of urticaria with acute localized œdema.

What is needed, in truth, is a dermatological Linnæus, to bring order out of the chaos at present existing in the group of erythemas. While I feel that in bringing together a somewhat motley series of cases I may only have contributed to make the "confusion worse confounded," on the other hand there is, I think, a positive advantage in recognizing the affinities and the strong points of similarity in affections usually grouped as separate diseases.

VENOUS THROMBOSIS AS A COMPLICATION OF CARDIAC DISEASE.

By WILLIAM H. WELCH, M.D.

ONE of the most interesting points of view from which to consider the subject of thrombosis is that of its association with different diseases. The study of such association is not only of clinical interest, but is capable of contributing to our knowledge of the causation of thrombosis, which in many respects is still obscure. Infectious and chronic wasting diseases are those most frequently complicated or followed by vascular thrombosis; typhoid fever and influenza heading the list among the former and tuberculosis and cancer among the latter. There are, however, a number of other diseases with which peripheral thrombosis may be associated more or less frequently, and to one of these less generally recognized associations I wish to call attention in this paper.

Although there are scattered reports of a number of instances of the occurrence of venous thrombosis in diseases of the heart, I cannot find that particular attention has been called to this complication either in text-books on these diseases or in special monographs. This is doubtless attributable mainly to the infrequency of the complication, perhaps also in part to a failure to recognize it. By far the largest number of cases have been reported by French physicians.

As will appear from the following reports of cases there are certain peculiarities of the venous thrombosis of heart disease which render this subject well worthy of investigation. For the clinical histories of the cases from the Johns Hopkins Hospital I am indebted to my colleague, Doctor Osler.

Case 1. — *Aortic ana mitral insufficiency. Adherent pericardium. Broken compensation. Thrombosis of left innominate, jugular, subclavian, and axillary veins. Death.*

R. H., negress, aged seventeen, admitted November 26, 1898, died January 16, 1899. Nothing of importance in family history or in personal history, until the occurrence, six years ago, of a severe attack of inflammatory rheumatism, with swelling and tenderness of most of the joints. Since then she has not been strong and has had at times rheumatic pains. In January, 1898, occurred a second attack of articular rheumatism, since which she has suffered

from shortness of breath on exertion, palpitation, pain in the region of the heart, and some cough. These symptoms became aggravated during the last two months.

Upon admission, patient, who is well nourished, is suffering from respiratory distress. No œdema of extremities. Pulse 112, somewhat irregular, with fair volume and tension. Respirations 40. Temperature 99.5° F. Large veins of neck full and pulsating. Marked bulging of precordium, and heaving impulse over heart, especially distinct below and outside of left nipple. Marked pulsation in episternal notch. Point of maximum intensity in sixth left interspace, 12 ctm. from midsternal line. Relative cardiac dulness begins above in left first intercostal space. Systolic thrill at apex, where on auscultation are heard intense musical systolic and rasping diastolic murmurs transmitted into axilla. In the aortic area both sounds are replaced by a loud to-and-fro murmur, the diastolic portion being especially rough. Second pulmonic sound intensely accentuated. Heart's action irregular and violent. Capillary pulse visible.

At apex of right lung, slight expansion, dulness, prolonged, almost tubular expiration. A few medium-sized moist râles at base of lungs. No tubercle bacilli in sputum.

Liver somewhat enlarged. Urine albuminous, with hyaline and granular casts. Blood count: red corpuscles, 4,524,000; leucocytes, 12,400; hæmoglobin, 65 per cent.

Patient improved somewhat after admission, but her general condition continued much the same. The pulse at times was very intermittent and there was much tenderness over the precordial area. The temperature varied from normal to 101° F.

January 14th: For the first time œdema of the left arm is noticed, most marked around the elbow joint. The right arm is free from swelling. The face and legs are moderately œdematous. Complains of severe headache. Pulse 120, irregular and weak.

January 16th: Œdema of left arm, which is painful, has increased. Temperature 100.5°. Pulse very intermittent. Death occurred rather suddenly at 6:30 P.M.

Autopsy (Dr. MacCallum) 14 hours after death: Body of a girl, rather slenderly built, 162 ctm. long. Moderate œdema of ankles and feet. Extensive œdema of left arm, especially about the elbows, the hand being but little swollen. No œdema of right arm and no definite swelling of face or neck.

About 200 c. c. clear yellowish fluid in peritoneal cavity. Surfaces smooth and glistening. Precordial space greatly enlarged, measuring 16 ctm. transversely and 10 ctm. vertically. Firm adhesions between parietal pericardium and the pleura, diaphragm, and other surrounding tissues. Left pleural cavity contains over 300 c. c. slightly turbid, yellowish fluid, the left lung being much compressed by this and the enlarged heart. There are many easily torn pleural adhesions. The right pleural cavity contains a small amount of fluid and presents a few light adhesions.

The pericardial sac is obliterated by fibrous adhesions. The heart weighs 720 grammes, and gives the following measurements: right ventricle 8 ctm. long, its wall 6 mm. thick; left ventricle 9½ ctm. long, its wall 16 mm. thick; tricuspid orifice 10½ ctm., mitral 10 ctm., aortic 7 ctm. All of the cavities much dilated. Left auricle greatly dilated, reaching nearly to first rib. Tricuspid and pulmonic valves delicate. Mitral orifice very wide; valvular segments, particularly the posterior, thickened and retracted. Aortic valvular segments much thickened, stiff, and curled at their edges, so as to be markedly shortened. No fresh vegetations. Coronary arteries patent and free from sclerosis. The aorta shows yellow streaks of fatty degeneration of the intima, and a few small elevated patches of fresh sclerosis.

The left innominate vein, the left internal and external jugular veins, the left subclavian and axillary veins are occluded by a continuous fresh thrombus mass. The prevailing color of the thrombus is dark reddish. The part occupying the innominate just before the reception of the jugular vein and that filling the well developed bulb of the internal jugular vein is gray or grayish-red, firmer and more adherent to the wall, therefore older, than the dark-red, softer clot in the distal portions. The thrombus in the jugulars stops somewhat below the level of the larynx. The brachial and other veins of the arm are free from thrombus.

The tissues around the thrombosed veins are œdematous, and freshly swollen lymphatic glands are present in their neighborhood. The corresponding veins of the right side are free from thrombus.

The lungs are dry, tough, and of a salmon-pink color, evidently the seat of chronic passive congestion. The bronchi contain blood-stained mucus. No areas of fresh consolidation. Pulmonic vessels free from thrombi.

The liver is moderately enlarged and presents typical nutmeg mottling. Spleen also enlarged, firm, dark-red, slightly adherent to surrounding tissues; Malpighian bodies distinct. Kidneys swollen, congested, the seat of chronic passive congestion. Gastric and intestinal mucosæ deeply congested, the solitary follicles swollen. No important changes in other organs.

Microscopical Examination: The swollen lymph glands show marked hyperplasia of the endothelial cells. The heart muscle is slightly fatty and shows scattered focal accumulations of small round cells, mainly of the lymphoid type. The kidneys show passive congestion and parenchymatous degenerations, without increase of the connective tissue. The lungs present the usual evidences of chronic passive congestion of moderate degree.

Sections of the thrombosed veins show that the grayish-red part of the thrombus is composed of platelets, fibrin, and leucocytes with entangled red corpuscles. A coral-like arrangement of the platelet masses is indicated, but is not very distinct. Leucocytes are fairly numerous. There are no evidences of organization. The intima and outer coats contain a few leucocytes, and the intima is somewhat swollen, but there are no signs of sclerosis or other chronic affection of the veins. The redder parts of the thrombus are richer in red corpuscles, but here also islands and bands of platelets and fibrillated fibrin are present. A few chains of streptococci are detected in sections stained by Gram's method.

Bacteriological Examination (Dr. Harris): Plate cultures on agar made with all necessary precautions from the thrombi in the jugular veins showed a considerable number of small, grayish colonies, which were demonstrated to be of Streptococcus pyogenes. No other organism appeared in the cultures. Streptococcus pyogenes was cultivated also from the lungs. Cultures from the heart's blood, œdematous tissue in left axilla, the spleen and other organs were sterile.

The principal points of interest in the preceding case are the following: A girl, seventeen years old, with chronic aortic and mitral endocarditis following acute articular rheumatism, and giving rise to insufficiency of both valves with relative insufficiency of the tricuspid valve, suffered from the effects of broken compensation. During the last days of life, painful œdema of the left arm made its appearance, without implication of the right arm. At the autopsy, in addition to the advanced cardiac lesions and their customary secondary effects, an infectious thrombus was found filling the innominate, subclavian, axillary, and lower parts of the jugular veins on the left side. The oldest part of the thrombus occupied the lower bulb of the internal jugular vein, and the adjacent part of the innominate vein. The micro-organism concerned was Streptococcus pyogenes, which was present in the lungs and the thrombus, but was not found elsewhere.

The three following cases are also from Doctor Osler's service in the Johns Hopkins Hospital.

Case 2.—Mitral stenosis. Thrombosis of left jugular, axillary, subclavian, and innominate veins. Embolism of left popliteal artery. Recovery from effects of vascular occlusion.

E. O., female, aged thirty-five, admitted January 4, 1899. History of rheumatism in family. Diphtheria at ten, chorea at eleven years of age, accompanied by paralysis of the right side. Since these attacks patient has not been strong. No history of scarlet-fever, pneumonia, nor typhoid fever. Obscure history of repeated attacks of "rheumatism" without definite articular symptoms. Patient has suffered for years from shortness of breath on exertion, indigestion, nervousness, and chronic invalidism. For three weeks before admission, has been in bed with epigastric pain and digestive disturbances.

Upon admission patient is very nervous. The point of maximum intensity of cardiac impulse cannot be detected by palpation or inspection, but by the stethoscope is located in the fifth intercostal space, 10 ctm. from median line. Area of cardiac dulness not much increased. Very distinct thrill can be felt at the apex, where is heard a rough, intense presystolic murmur terminating in a short, sharp first sound. No second sound is heard at the apex. Over the body of the heart the valvular sounds are distinct and snapping. The second pulmonic sound is markedly accentuated. Pulse 144, small, irregular both in force and rhythm. Respirations 30. Temperature 99° F.

Medium-sized moist râles are heard behind, over the lower parts of both lungs, where there is also some impairment of resonance.

The absolute hepatic dulness extends from the seventh rib to a point 5½ ctm. below the costal margin in the mammillary line. The border of the liver can be distinctly felt. There is fairly distinct pulsation of the liver. The epigastric and right hypochondriac regions are somewhat tender to pressure.

There is a slight œdema of both ankles. Urine, sp. gr. 1.017, contains a small amount of albumin and hyaline and granular casts.

January 12th: The left ankle-joint is swollen, red, and tender.

January 19th: The left side of the neck is swollen, and painful upon pressure or movement. Temperature 100°. Pulse 124. Respirations 32.

January 20th: The fulness and tenderness of the left side of the neck have increased and a sensitive, cord-like body can be felt in the course of the internal jugular vein in its lower part, indicative of thrombosis. The pain and swelling of the left ankle-joint have disappeared. There are evidences of partial consolidation of the right lung below and behind. Moist râles are heard also at the angle of the left scapula. Patient is flighty.

January 26th: To-day appeared an œdematous swelling of the left arm, extending to the hand. The left arm measures 2 ctm. more than the right just above the wrist.

January 27th: The œdematous, painful swelling is now very marked, and occupies the whole of the left side of the neck, the pectoral region on the left side, the left shoulder, and the left arm to the hand. The œdema has a brawny, indurated character, but there is pitting on pressure. There is no marked difference in the superficial temperature of the two arms. The superficial veins of the arm and neck are distended. On account of the œdema the deeper veins cannot be distinctly palpated. Temperature 101°. Pulse 112. Respirations 40.

January 31st: The swelling of the left arm is less, that of the neck continues.

February 9th: A cord-like swelling of the external jugular vein can be traced up to the angle of the jaw. The axillary vein is likewise thrombosed.

February 26th: The hard œdema of the left arm and neck has continued, at times lessening and then returning. There is œdema of both lower extremities.

March 16th: Pain in the left side and a marked pleuritic friction rub on auscultation.

Soon after this date the general condition of the patient improved and the œdema of the arm and neck gradually disappeared through the establishment of a collateral circulation.

On November 24th embolism of the left popliteal artery occurred, characterized by loss of pulsation in left popliteal and tibial arteries, sudden pain, numbness, cyanosis, and coldness of the left foot and leg. The history of this embolic attack need not be given in detail. Suffice it to say that a collateral circulation was completely re-established. The patient is still in the hospital.

The diagnosis made by Doctor Osler in this case was mitral stenosis with thrombosis of the left innominate, left internal and external jugulars, left subclavian, and left axillary veins. The location, extent, and persistence of the hard, painful œdema, lasting for nearly three months, make probable the existence of thrombosis of the left innominate and subclavian veins, while that of the jugulars and axillary vein was definitely recognized. The history indicates that the thrombus started in the lower part of the internal jugular vein. It is to be noted that the onset of the thrombosis was preceded for a few days by a red, painful swelling of the left ankle-joint and was accompanied by evidences of acute pneumonia and by elevation of temperature, also that during its course acute pleurisy appeared. There is, therefore, much probability in the supposition that the thrombus in this case, as in the preceding, was of infectious nature.

Case 3.— *Mitral and aortic insufficiency. General anasarca. Thrombosis of left axillary and brachial veins. Death.*

H. M., male, aged sixteen, admitted February 24, 1900; died March 8, 1900.

No history of infectious disease except pneumonia at seven years of age. Present illness began in August, 1899, with vomiting and indigestion. Says that he had rheumatism in September, but no definite history of affection of joints was obtained. Repeated attacks of gastric pain and vomiting during the autumn. In January, feet, legs, and abdomen became swollen and he was confined to bed. The dropsy increased and a week ago the face and hands began to swell. Continual shortness of breath. Digestive disturbances continued.

Examination on Admission: Patient is propped up in bed; respirations 32, somewhat labored and irregular. Cyanosis of face and extremities. Œdema of face, thorax, upper and lower extremities, penis, and scrotum. Marked œdema of left forearm and hands. Ascites. Pulse 124, regular in force and rhythm, fair volume, low tension, hyperdicrotic. Pulsation of cervical veins.

Distinct precordial bulging and general heaving in this region. Apex beat in fifth interspace 11 ctm. to left of median line. Area of cardiac dulness increased, extending 1 ctm. to right of sternum and upward to left second rib. At apex first sound replaced by loud systolic murmur heard far out in axilla; second sound faint. In aortic area both sounds enfeebled, the second sound being accompanied by a faint diastolic murmur traceable down along right border of sternum. Second pulmonic sound distinctly accentuated.

Physical signs of a moderate amount of fluid in left pleural cavity are present, also some dulness and impairment of respiratory and vocal sounds on right side below and behind with a few fine moist râles.

Absolute hepatic dulness begins at sixth rib and is continuous with the abdominal flatness due to ascites, which is marked.

The œdema of the left arm and hand is so much greater than that of the right arm that thrombosis was suspected. Upon palpation the left axillary and brachial veins can be distinctly felt as hard, swollen, somewhat sensitive, cord-like cylinders, which can be made to roll beneath the finger.

Blood Count: Red-blood corpuscles, 6,900,000; leucocytes, 14,600; hæmoglobin 76 per cent.; 86 per cent. of the leucocytes are polymorphonuclear. Urine contains a small amount of albumin and some hyaline casts; sp. gr. 1.027.

After admission, the œdema of the left arm and hand continued to increase and reached an extreme degree so that splits appeared in the corium. The superficial veins were

distended. Temperature most of the time a little below normal, only occasionally rising to 99° and once to 100° F.

March 1st: Jaundice appeared. The next day a hæmorrhagic eruption appeared over the abdomen. Cheyne-Stokes breathing set in, the sputum became tinged with blood, and death occurred March 8th. Unfortunately permission could not be obtained for an autopsy.

In the preceding case of uncompensated mitral regurgitation the general dropsy was so great that only the excess of œdematous swelling of the left arm led to examination for venous thrombosis, of which positive evidences were found in the left brachial and axillary veins. Whether other veins were also implicated could not be determined, as no autopsy was permitted.

Case 4.—Mitral insufficiency. Thrombosis of left femoral vein. Recovery from effects of thrombosis.

M. H., male, aged seventy-eight, admitted December 27, 1898; discharged January 10, 1899. Patient had been in hospital a year ago suffering from abdominal pain and constipation. At this time mitral insufficiency was recognized. No history of rheumatism or of other infectious disease, except measles and smallpox in childhood. Has suffered of late years from pain in the abdomen, constipation, bronchitis, and increased frequency of urination.

Three days before admission was seized with pain on inner side of left ankle and inside of upper part of thigh, soon followed by swelling of the left leg and about the ankle.

On admission a rough systolic murmur is heard at the apex, transmitted to the axilla, and heard also over the body of the heart. Systolic whiff over aortic and pulmonic areas. Point of maximum intensity of cardiac impulse in fifth intercostal space 11 ctm. to left of median line. Veins of neck full and heaving, but without distinct pulsation. Superficial veins of nose and cheeks dilated. Physical signs of emphysema and bronchitis. Pulse 96, regular. Respirations 20. Radial and temporal arteries tortuous and sclerotic.

The left lower extremity œdematous from the groin to the foot, the swelling being most marked around the ankle. Superficial temperature of left leg somewhat higher than that of right. Superficial veins dilated. An indurated, sensitive cord can be felt in the left Scarpa's triangle running obliquely downward and inward. The deep lymphatic glands below Poupart's ligament on the left side are somewhat enlarged. The pulsation of the left femoral artery is less distinct than that of the right. Slight œdema of the right leg.

Blood Count: Red-blood corpuscles, 3,800,000; leucocytes, 5000; hæmoglobin, 40 per cent. Temperature remained about normal, occasionally rising in the evening to a little over 99° F. Urine: specific gravity, 1.020; acid, faint traces of abumin; no casts.

The œdema of the left leg gradually lessened, and on January 10th patient was discharged at his request with only a little œdema of the extremities.

In the foregoing case there were no marked evidences of disturbed compensation of the mitral lesion, and there was arterial sclerosis. The relationship of the venous thrombosis to the cardiac lesion was not, therefore, so evident as in the three cases first reported, and probably this case does not properly belong in the same category.

Thrombosis of pulmonary vessels, which is not particularly uncommon in uncompensated cardiac disease, is of course not to be included in the same class as peripheral venous thromboses. I have also excluded

from consideration the venous thromboses complicating general arterio-sclerosis and chronic nephritis even when associated with cardiac hypertrophy and dilatation, although it is possible that some of these might appropriately have been included. In general, however, they belong to a different category. Our autopsy records contain five cases of thrombosis of the femoral and iliac veins complicating arterio-sclerosis and chronic nephritis accompanied by cardiac hypertrophy. In two of these there was atheroma of the aortic valves with some insufficiency. There is one instance of thrombosis of the superior mesenteric veins associated with small kidneys and cardiac hypertrophy. A case of thrombosis of the iliac and femoral veins on both sides associated with pulmonary emphysema and hypertrophy and dilatation of the right side of the heart has also been omitted as not falling under the class considered in this article. For the same reason I have omitted a case in our records of thrombosis of the right femoral vein complicating cirrhosis of the liver with sclerosis, calcification, and insufficiency of the aortic valves, and also instances of thrombi in varicose veins in cases of heart disease.

The arterial thromboses complicating cardiac diseases are of much interest. Doubtless most of those in the systemic arteries are of embolic origin, but there is evidence that some, particularly in cases of extreme mitral stenosis, are autochthonous. This subject, to which I have given some attention in my article on "Thrombosis and Embolism" in *Allbutt's System of Medicine*, does not fall within the scope of the present paper.

The only attempts, which I have been able to find, to collect from the records cases of peripheral venous thrombosis complicating diseases of the heart are those by Parmentier (1889),[1] by Hirschlaff (1893), and by Kahn (1896). Parmentier and Kahn each report a single case from Hanot's clinic and refer to four others which they say are the only ones they can find in French literature. Hirschlaff adds to Parmentier's list two personal observations and the two cases of Robert (1880) and of Ormerod (1889). Peter, in 1873, in his *Leçons de clinique médicale*, reports a case and devotes a part of one chapter to an interesting consideration of venous thrombosis in diseases of the heart without adding further observations. Huchard's article (1897), "Cachexie cardiaque et thromboses veineuses," and Helen Baldwin's report of a typical case (1897), the only one which I have met in American literature, deserve especial mention. Without pretence to completeness I have been able to collect reports of twenty-three cases to be added to the four personal observations already described. I have little doubt that

[1] The references will be found at the end of this article.

a more diligent search would bring to light other reported cases. I shall present in chronological order abstracts of these twenty-three cases, of some of which the histories are very meagre.

Case 5.—Bouchut (1845). Woman with heart disease succumbed to obliteration of the deep and superficial veins of the left leg. No further details.

Case 6.—Bouchut (1845). Man with heart disease, in whom occurred obliteration of the superior vena cava, jugular, axillary, and deep arm veins. Engorged, painful, hard, venous cords could be felt in neck and arms. Cyanosis and œdema of face, neck, and arms. Innumerable varicose, agglomerated, large veins appeared in neck and over thorax. A satisfactory collateral circulation developed, and the man left the hospital free from cyanosis and œdema.

Case 7.—Cohn (1860). Woman, sixty-one years old. Stenosis and insufficiency of aortic valves, some thickening of mitral segments, cardiac hypertrophy, and symptoms of non-compensation. For fourteen weeks œdema of left foot and leg, later also of right foot, but here less marked. At autopsy, adherent old thrombi in both iliac veins, extending on the left side half-way down the thigh, and into some of the larger tributaries of the femoral vein.

(Cohn reports two other cases of thrombosis of veins of the lower extremities with cardiac disease, but in these there was general arterial sclerosis and no pronounced valvular lesion.)

Case 8.—Jolly (1861). Woman with organic disease of the heart had phlegmasia alba dolens of both upper extremities. Death occurred from an ulcero-membranous affection of the intestine. No further details.

Case 9.—Ramirez (1867). Boy, aged twelve. Acute articular rheumatism one year before admission. Aortic and mitral insufficiency, general anasarca, dyspnœa, and other symptoms of extreme cardiac incompetence. Bloody expectoration. Thrombosis of jugular, axillary, and subclavian veins on the right side. The obliterated external jugular vein very evident. Painful œdema of right side of neck and right arm. Death in coma. No autopsy.

Case 10.—Ramirez (1867). Man, aged fifty. For six months following intermittent fever, dyspnœa, general œdema. Incompletely compensated aortic and mitral insufficiency. Œdema of lower extremities almost disappeared, while that of face and upper extremities persisted. Dyspnœa increased. Painful, hard œdema of left arm and left side of neck appeared, and two large, hard, sensitive cords, formed by the obliterated jugular veins, could be felt.

Autopsy showed thickening and retraction of aortic valves, with relative mitral insufficiency. Great dilatation of all cardiac cavities. Blackish, firm thrombi in left internal and external jugular, subclavian, and axillary veins.

Case 11.—Duguet (1872). Patient, who formerly had acute articular rheumatism, was admitted for dyspnœa, cyanosis, palpitations, and œdema of lower extremities. Three days before death there appeared painful, hard œdema of left arm, and the axillary vein was felt as a hard cord. Autopsy showed marked stenosis, with insufficiency of the mitral orifice, without fresh endocarditis, with cardiac hypertrophy and dilatation, hydrothorax and hydropericardium, pulmonary infarcts, and grayish-black, non-adherent thrombi filling the left subclavian, axillary, and brachial veins.

Case 12.—Peter (1873). Woman with mitral insufficiency and obstruction, and with relative insufficiency of the tricuspid valve, suffered from œdema of lower extremities, ascites dyspnœa, cyanosis, and great congestion of lungs and liver. For past ten days left arm œdematous throughout, with tenderness at certain points ; later outer and lower part of neck became swollen and painful, first near the junction of jugular and subclavian veins. The obliterated subclavian [axillary ?] and external jugular veins could be felt as hard, cylindrical cords. Venous pulse, formerly present in cervical veins of both sides, is now evident only

on the right side. The left radial pulse is almost imperceptible from pressure of thrombosed veins on the arteries. At the end of three weeks collateral circulation was established and œdema of arm had disappeared. General condition otherwise unimproved.

Case 13.—Robert (1880). Woman, age between thirty and forty years, suffered for eight years with cardiac symptoms following pneumonia, of late palpitation, dyspnœa, cyanosis, œdema of lower extremities and abdominal wall, moderate ascites, pulsation of veins of neck, bronchitis, and pulmonary congestion. Mitral stenosis and relative tricuspid insufficiency diagnosed. For over a month before death patient had increasing œdema of left arm (right being free) and slight tumefaction of left side of face. Left internal jugular vein felt as hard cord. Veins over left pectoral region became distended.

Autopsy: Extreme mitral stenosis, with hypertrophy and dilatation of the right heart, double hydrothorax, pulmonary infarcts. Thrombosis of left innominate, subclavian, and both jugular veins. The left innominate was notably narrowed at confluence of jugular and subclavian veins, and here and in the lower end of the jugular was a firm, fibroid, adherent thrombus, evidently the oldest part of the clot. The thrombus elsewhere was in places centrally softened. The walls of the left subclavian vein were thickened. The clot in the jugular could be traced to the entrance of the linguo-facial trunk. Lymphatic glands in neighborhood of thrombosed veins are enlarged. Superior vena cava free.

Case 14.—Parmentier (from Hanot's clinic) (1889). Woman, aged forty. For four years, following puerperal infection, cardiac symptoms; for last five months, palpitation, dyspnœa, cough, œdema of lower extremities; and for three weeks before admission painful œdema of left arm and corresponding mammary region, later slight œdema of right arm. Upon admission: very rapid, irregular heart's action; irregular, small, intermittent pulse; marked venous pulsation from systolic reflux, intense dyspnœa, pulmonary râles, and albuminuria. The left upper extremity is tumefied throughout its whole extent by a hard, tender œdema, which extends also to the left mammary region. Symptoms increased in gravity and death occurred four days after admission.

Autopsy: Extreme mitral insufficiency and dilatation of all cavities of heart, left hydrothorax, ascites, nutmeg liver. Left subclavian vein and small communicating veins completely obliterated by a reddish thrombus, grayish and most adherent in the middle of the vein, where it is evidently oldest, and where there is partial organization. Upon microscopical examination the coats of the vein were found thickened and the capillaries in outer coats dilated. The jugular veins were free and no thrombus was found in other veins of the extremities.

Case 15.—Ormerod (1889). Patient was admitted for mitral stenosis. At autopsy was found complete obstruction by adherent clot of both innominate veins, internal jugulars, subclavians, anterior and external jugulars, the clot being firmer on right than left side. A projecting end of clot hung into superior cava, but was not adherent there and ceased at the opening of the azygos. Left auricle and right cavities of heart much dilated. The symptoms of thrombosis, which developed in the hospital, pointed to its commencement in right subclavian vein. No local cause for it could be found. Specimens were presented before the London Pathological Society and no further details are given.

Case 16.—Hirschlaff (from Leyden's clinic) (1893). Woman. Repeated attacks during last ten years of articular rheumatism. Cardiac symptoms existed for some time before admission. Upon admission, respirations hurried and labored, œdema of lower extremities, ascites, moist pulmonary râles behind and below, heart's action violent, with diffuse precordial heaving, cardiac dulness much increased in all directions, loud systolic and diastolic murmurs at apex, feebler systolic murmur with indistinct diastolic sound in aortic area, distinctly visible venous pulsation, pulse 120, of low tension, enlargement of liver and spleen, moderate albuminuria, diminished excretion of urine. No material improvement followed. Both hands became œdematous. Six days before death there appeared œdema of the left half

of the thorax, reaching the middle line and also of the left supraclavicular fossa, later œdema of left half of face. Pulsation disappeared from the left external jugular, while it persisted in the right. A painful hard cord could be felt in the left supraclavicular fossa extending upward along the course of the external jugular vein, and a similar one along that of the internal jugular. Erysipelas of the right leg set in after insertion of Ziehl's needles to relieve the œdema, and patient died in collapse.

Autopsy: Extreme stenosis and marked insufficiency of the mitral valve and slighter insufficiency of the aortic valve. Cavities much dilated. Thrombosis of the entire left innominate, internal jugular and smaller tributaries, external jugular, subclavian, and axillary veins. The thrombus is reddish yellow and oldest in the jugular bulb. The veins of the upper arm contained fluid blood. Cerebral sinuses free. Red induration of both lungs. Small quantities of fluid in both pleural cavities. Fresh hyperplasia of spleen.

Case 17.—Hirschlaff (1893). Woman, aged fifty-one. Diphtheria at eighteen, articular rheumatism, without evident sequels, at twenty years of age. For past twenty years, epilepsy with periodical swelling of both legs, lasting for months. For past two years, persistent œdema of legs and for a month before admission palpitation and dyspnœa. Shortly before admission, increasing painful swelling of left side of neck and left arm. On admission, marked œdema of lower extremities and of the left side of neck and left arm, slight ascites, slight cyanosis and icterus, systolic pulsation of left external jugular, enlargement of heart, heaving impulse and thrill at apex, loud murmur filling entire period before systole at apex, diastolic murmur in aortic area, dulness and crepitant râles over posterior, lower chest; urine diminished, concentrated, and slightly albuminous; pulse 108, small, irregular. The veins in the left side of neck and over shoulder became much distended, and the œdema increased in extent. Three weeks later the right side of the neck and thorax and then the face became œdematous. Pulmonary symptoms. Repeated epileptic attacks occurred, accompanied and followed by extreme cyanosis and dyspnœa. In one of these death ensued a month after admission.

Autopsy: Cardiac hypertrophy, extreme dilatation of right ventricle and auricles, tricuspid valve thickened, its chordæ shortened and grown together, extreme stenosis of mitral orifice, insufficiency of aortic valves, on which are fresh vegetations; myocardium flabby, pale, and yellowish. Moderate accumulation of fluid in pleural and pericardial cavities; recent and old pulmonary infarcts; chronic passive congestion of liver, spleen, and kidneys. Thrombosis of superior vena cava, left innominate vein, left subclavian and both internal jugular veins. The oldest part of the thrombus occupies the bulb of the left jugular, where it is firm, closely adherent, and partly organized. Tissues around thrombosed veins behind manubrium sterni œdematous. Small thrombus in the left lateral sinus of the dura mater.

Case 18.—Gatay (1895). Woman, aged twenty-eight. Ten years ago acute rheumatism and endocarditis, followed by palpitation and articular pains. A few days before admission patient was seized with chills followed by dyspnœa. On admission cyanosis, œdema of lower extremities, areas of pulmonary consolidation, disorderly action of heart, pulse small and intermittent, albuminuria, temperature 39.8° C. Four days later hard œdema of left arm, most marked in lower part, appeared. Fluid in wrist joint. Hard venous cord on inner side of left upper arm was felt.

Autopsy: Much hypertrophy and dilatation of heart, the right ventricle markedly dilated and tricuspid valve relatively insufficient, mitral valve thickened, retracted, and incompetent, old fibrous plaques on endocardium, no fresh endocarditis. Pulmonary infarcts. Left brachial vein swollen and filled with a red, adherent, centrally softened thrombus, 10 ctm. long. Venous wall above and below thrombus red and thickened.

Case 19.—Kahn (from Hanot's clinic) (1896). Woman, aged fifty-two. Seven years ago, influenza followed by some œdema of legs, which soon disappeared. For four months before admission, cough, weakness, œdema of legs, loss of flesh. On admission, cardiac hypertrophy,

systolic murmur at apex transmitted into axilla, no murmur at base, pulse 100, small, soft, radials sclerotic, external jugulars turgid but without pulsation, respirations rapid and labored, œdema of lower extremities, emaciation. Ten days after admission painless œdema of right hand and forearm appeared. Temperature 38.3° C. Subcrepitant râles at base of lungs. The œdema rapidly spread so as to involve whole of right arm and corresponding mammary region. There was no pain. The skin was colder than on the left side and somewhat mottled with violet patches. Dyspnœa intense, respirations 42, urine albuminous, sputum bloody, temperature 39° C. ; delirium and death in coma about three weeks after onset of thrombosis.

Autopsy : Hypertrophy of whole heart, right cavities much dilated, mitral valve indurated, thickened, retracted, insufficient without stenosis ; other valves normal, fibrous myocarditis. Fluid in left pleura, pulmonary infarcts, chronic passive congestion of liver and kidneys, spleen swollen and soft. There are five or six enlarged glands near the right subclavian vein which is completely filled with a reddish, fibrinous, firm, non-adherent thrombus, 4 ctm. long, extending to the origin of the axillary vein. No thrombi in collateral veins. On microscopical examination, coats of thrombosed vein appear thickened, and capillaries in outer wall dilated.

Case 20.—Mader (1897). Woman, aged forty-five. Repeated attacks of articular rheumatism followed by palpitation, dyspnœa, and œdema of lower extremities. On admission evidences of stenosis and insufficiency of the mitral and tricuspid valves. Marked ascites. Three weeks later patient complained of great tension in veins of arms, and in fact these became enormously distended, as did also the cervical and upper thoracic veins. Collateral veins could be traced to the epigastric veins. There was vertigo, and the lips became cyanosed. With the establishment of a collateral circulation there was gradual improvement in the symptoms. The diagnosis was thrombosis of both innominate veins, possibly also of superior vena cava, complicating the valvular affections mentioned.

Case 21.—Helen Baldwin (1897). Girl, aged nineteen. Three attacks of rheumatic fever, the last seven years ago. Five weeks ago œdema of the feet. A few days before admission, began to suffer from pain in left axilla and left side of neck, also from swelling of the left side of neck. The latter at first would disappear in the erect posture. Amenorrhœa. On admission, great weakness, œdema of both legs, cyanosis, dyspnœa, respirations 40, pupils dilated ; pulse 108, small, rapid, and compressible ; marked enlargement of heart, double aortic and mitral murmurs, ascites, enlargement of liver, albuminuria, granular and hyaline casts, quantity of urine diminished, sp. gr. 1.033. On left side of neck is a hard, tender swelling of lower part of external jugular vein, which is tortuous. There is a hard cord in left axilla extending for about five inches down the arm. Left arm œdematous and pale. The next day external jugular vein was felt as a cord up to one half inch of lobule of ear. Œdema of whole left side of neck. Signs of beginning double pneumonia. For five days there was noted a persistently low temperature when measured by thermometer in mouth, whereas that by the rectum was 6 to 7.8 degrees higher. Bloody expectoration on the fifth day. Death six days after admission.

Autopsy : General œdema, ascites, double hydrothorax, marked hypertrophy of heart, the auricles extremely dilated, chronic changes in all of the valves, the most marked being stenosis and insufficiency of the mitral valve, the edges of tricuspid valve thickened and curled. Pulmonary congestion and infarcts and early stage of double lobar pneumonia. Chronic passive congestion of abdominal viscera. Firm thrombi fill the left innominate, subclavian, external and internal jugular veins, extending nearly to lobe of ear. Veins of extremities could not be further examined. Enlarged lymph nodes along trachea, not pressing on veins. Small extravasation of blood along left side of trachea and behind the left auricle. On microscopical examination the thrombi were found to be partly organized. There was marked chronic thickening of the intima of the subclavian vein, which was believed to be the starting-point of the thrombus.

Case 22.—Huchard (1897). Woman, aged twenty-four. Seven years ago had articular rheumatism with cardiac complication, followed by mitral stenosis. Painful swelling of left arm appeared during convalescence from an undetermined febrile disease. On admission severe dyspnœa; pulse rapid, small, irregular; expectoration bloody, urine slightly albuminous, no œdema of lower extremities. Painful, hard œdema of entire left arm with prominence of superficial veins, and hard, sensitive, voluminous, venous cords. Death in coma nine days after admission.

Autopsy: Double hydrothorax, pulmonary infarcts, slight ascites, nutmeg liver. Heart weighed 450 grm.; buttonhole mitral orifice causing extreme stenosis, slight narrowing of tricuspid orifice, and marked dilatation of right auricle. Large stratified thrombus in left auricle. A few pericardial adhesions. Left brachial vein markedly dilated, and filled in upper part with an adherent thrombus 3 ctm. long, starting from a valvular pocket.

Case 23.—Huchard (1897). Male with mitral stenosis and thrombosis of veins of left lower extremity. No other details.

Case 24.—Nicolle and Robineau (1897). Girl, aged sixteen and a half. Repeated attacks of rheumatism during last four years. Chief symptom on admission, respiratory distress; no œdema. Four months after admission, patient felt pain in the left side of neck and head, and a hard, painful cord, 5 to 6 ctm. long, was detected in the course of the left external jugular vein. Three days later the thrombus had extended and painful œdema of left side of neck and face appeared. Superficial veins dilated; urine slightly albuminous. Ten days after first symptoms of thrombosis, entire left upper extremity became œdematous. Expectoration bloody. Œdema increased, and appeared also in lower extremities. Death occurred a little less than a month after the beginning of the thrombosis.

Autopsy: Mitral orifice narrowed, numerous hard, papillary vegetations on borders and surface of mitral segments, also on the aortic valves and the free borders of the tricuspid. Chordæ tendineæ of mitral valve shortened. Hydrothorax, many pulmonary infarcts, pneumonia of left lower lobe. Thrombosis of left external jugular vein and branches and of subclavian vein. Thrombus black, rather soft, slightly adherent, unorganized. Venous walls, especially inner coat, thickened. Swollen, hæmorrhagic lymph glands surround the thrombosed veins. Tissues of neck very œdematous.

Case 25.—Poynton (from service of Dr. Lees), 1898. Girl, aged nineteen. Very severe attack of rheumatic fever, lasting thirteen weeks, nine months before admission. Swelling of legs and abdomen noticed two months before admission, and a few days before admission left arm suddenly began to swell. On admission, great pallor and distress, evidences of advanced organic disease of heart, systolic apical murmur, accentuated second pulmonic sound; pulse 90, irregular in force and rhythm; œdema of legs and thighs, of upper extremities and upper part of chest, the left arm and hand being very much swollen; impairment of resonance and respiratory sounds over bases of lungs, liver large and tender, conjunctivæ icteric, urine somewhat albuminous, temperature subnormal and continuing so throughout illness. Death, preceded by cerebral symptoms and coma, about two weeks after admission.

Autopsy: Totally adherent pericardium, evidences of old rheumatic endocarditis, aortic, mitral, and tricuspid valves incompetent, and mitral orifice slightly narrowed; cavities dilated, especially right ventricle; weight of heart, 16 ounces. Pleuræ adherent. Chronic passive congestion of viscera. Adherent firm thrombi filled both innominate and both internal jugular veins, the lower end of left internal jugular being white, narrowed, and very firm. Adherent mural thrombus in upper part of superior cava. Mediastinal tissues œdematous. Brain generally soft and œdematous.

Case 26.—Poynton (from service of Dr. Cheadle), 1898. Woman, aged twenty-one. In childhood scarlet-fever, followed by rheumatic fever. Two years ago second attack of rheumatism. Present illness began gradually with weakness, dyspnœa, and œdema of legs and face. On admission, anæmia, œdema of legs, face puffy, bronchitic sounds, harassing

cough, systolic thrill, loud systolic murmur over front and back, dilatation of heart. Temperature 101° F. ; respirations 28 ; pulse 128, of low tension. Urine albuminous, sp. gr. 1.020, contained blood and casts. Liver and spleen enlarged. Irregular pyrexia and sweating continued throughout illness in hospital. Twelve days after admission, pain in wrists and along inside of left forearm. Œdema of face, which had almost disappeared, returned, especially on left side. Irregularity of pulse, orthopnœa, symptoms of renal infarction and pneumonia appeared, and death occurred five and a half weeks after admission

Autopsy : Heart weighed 14 ounces, all its cavities much dilated ; both ventricles and left auricle hypertrophied ; muscle pale ; mitral orifice widened ; numerous exuberant vegetations of mitral segments, also on endocardium of left auricle and left ventricle ; chordæ tendineæ ulcerated through ; other valves normal, except slight widening of tricuspid orifice. Pulmonary congestion ; pneumonia of left lower lobe ; chronic passive congestion of abdominal viscera, with infarcts in spleen and kidneys (microscopically no evidence of interstitial inflammation or chronic nephritis). Left internal jugular, from junction with subclavian vein to angle of jaw, occluded by a thrombus, which was pale and adherent to wall, especially in lower part, where vein was cord-like. Innominate vein and right jugular not thrombosed. Brain normal. On microscopical examination no micro-organisms found in cardiac vegetations or in thrombus. Diplococci, staining by Gram, in pneumonic area. Sections of left internal jugular showed organizing thrombus, without distinct thickening of venous wall. Walls of right jugular normal. (No mention of cultures.)

Case 27.—Poynton (from service of Dr. Cheadle), 1898. Girl, aged nine. Two years ago scarlet-fever, since which heart was affected. For three weeks before admission, thoracic pain and cough, and for one week dropsy. On admission, February 19th, pallor, with some cyanosis ; orthopnœa ; fingers clubbed ; œdema of lower extremities, anterior chest wall and face ; some ascites ; marked increase of cardiac dulness to right and left ; precordial bulging ; epigastric pulsation ; systolic and diastolic thrill ; double mitral and aortic murmurs ; doubtful pericardial friction rub ; impaired resonance and respiratory sounds over bases of lungs ; scattered bronchitic râles ; liver enlarged and pulsating. Temperature 97.4° F. ; respirations 30 ; pulse 100, irregular, weak, and small ; urine, 1.025, contained albumin and blood, without casts. Improvement until March 10th, when pleural friction was heard in left axilla and dulness at left base, without rise of temperature. Again improvement until April 10th, when pulmonary symptoms reappeared and swelling of axillary glands was noticed. Heart more dilated, pulse feebler. 13th : left side of face swollen. 16th : marked œdema of right side of neck, eyelids, and lips ; face purple ; both sides of neck tense, painful to touch and on movement ; temperature subnormal. 17th : right arm began to swell, shortly afterward œdema of left arm and chest ; patient apathetic. 18th : drowsiness, cyanosis, sudden dyspnœa, with symptoms of collapse. 19th : tender œdema of right arm extreme. 20th : two firm cords felt in lower part of neck ; legs and feet a little swollen ; area of cardiac dulness enormous ; loud pericardial friction, crepitation over both lungs ; urine scanty, slightly albuminous, no blood. Death April 21st.

Autopsy : Acute sero-fibrinous pericarditis ; all cardiac cavities dilated and hypertrophied, the right relatively more than the left. Mitral and tricuspid valves incompetent ; fresh vegetations on aortic, mitral, and tricuspid valves. Usual visceral changes secondary to advanced cardiac disease. Obliterative thrombosis of superior vena cava in its upper two-thirds, both innominate, subclavian, internal and external jugular, and axillary veins, and the left inferior thyroid vein. Small adherent mural thrombus in left brachial vein. The oldest parts of the thrombus were in the lowest portions of the internal jugular veins and the left innominate, which were white, small, and firm, and adherent to surrounding tissues. Beyond these older thrombi the vessels were bulged with soft clot. The thrombus in the superior cava was soft, pale, non-adherent, except in its upper part.

Cultures and films from pericardial exudate and blood-clot negative ; sections of soft clot in superior cava and of thrombus in right axillary showed no micro-organisms. Thrombi

were undergoing organization in older parts. Early phlebo-sclerosis, apparently secondary to thrombus, in older thrombosed vessels. Venous wall not thickened where thrombus was fresh and not adherent. Interstitial myocarditis in subpericardial layers and beneath inflamed endocardium.

The most remarkable feature of the foregoing twenty-seven cases of venous thrombosis in heart disease, is the location of the thrombi: twenty-three were thromboses of veins conveying blood from the upper extremities or the neck, or both, mostly of the left side, and only four were thromboses of veins supplying the lower extremities. I do not suppose that these numbers represent the correct ratio between upper and lower venous thrombosis in heart disease, for thrombosis of veins of the lower extremities in this condition is much more likely to be overlooked in consequence of the more common and greater œdema of the lower limbs, and is also much less likely to be reported. How often it is overlooked can be at present only a matter of conjecture, but it is not probable that such thrombosis is at all frequent. In fact the comparative infrequency of peripheral venous thrombosis in cardiac disease is in itself a matter of interest, in view of the slow, feeble, and irregular venous circulation, and of the frequency of so-called marantic thrombi in the heart itself during the failure of compensation. The relatively small liability to venous thrombosis under such conditions of the circulation is one of many evidences that mere slowing of the blood current is not an efficient cause of thrombosis.

Even allowing for a considerable increase in the number of instances of thrombosis of the veins of the lower extremities as the result of more thorough search in cases of heart disease, the relatively large number of observations of thrombosis of the upper veins revealed in my collection of cases still remains most remarkable. Bouchut places the ratio of venous thromboses of the upper extremity to those of the lower at one to fifty. Of sixty-seven cases of peripheral venous thrombosis in our autopsy records at the Johns Hopkins Hospital, only one was of the upper extremities, although several instances of the latter have been observed clinically in the Hospital. Moreover, the four instances above recorded of venous thrombosis of the lower extremities in heart disease, had little in common with the remaining cases. Two were in old persons, with some arterial atheroma, and of two no satisfactory history is given. The thrombosis was on the left side in three and bilateral in one, thus conforming to the rule. As will appear from an analysis of the remaining twenty-three cases, the venous thromboses of the neck and arms in cardiac disease constitute a separate and distinct group, characterized by special features of unusual interest.

ANALYSIS OF TWENTY-THREE CASES OF THROMBOSIS OF VEINS OF NECK, ARMS, AND CHEST IN HEART DISEASE.

Sex.—Seventeen cases were of females, four of males, and of two the sex is not stated. While the total number of cases is too small to warrant percentage estimates, there can be no doubt that females are much more disposed to this form of thrombosis than males. Whether this disposition is more than an expression of the greater liability of females to the mitral lesions present at the ages in the cases observed, I must leave an open question. The figures seem somewhat out of proportion to this greater tendency.

Age.—One patient was nine years old, 6 were between ten and twenty, 3 between twenty and thirty, 3 between thirty and forty, 2 between forty and fifty, 2 between fifty and sixty, and of 6 the age is not stated. Of the 17 patients with thromboses of the neck and arms whose ages are stated, nearly one half (8) were between fifteen and thirty years of age. The youngest was nine and the oldest fifty-two.

Valvular Lesions.—In 2 cases the exact nature of the valvular lesion is not stated. Of the remaining 21 cases, there was organic disease of the mitral valve in 20; in the exceptional case there was aortic insufficiency with relative mitral incompetence.

The mitral lesion is described as insufficiency in 8 cases, as stenosis in 6, and as stenosis and insufficiency in 6. In the last group there were at least 3 instances of extreme stenosis. The aortic valves were affected in 10 cases, in all of these there being incompetence, somtimes also with obstruction.

The organic valvular lesion was limited to the mitral valve in 11 cases, to the aortic valve in 1, to the mitral and aortic valves in 6, to the mitral, aortic, and tricuspid valves in 2, and in 1 case all of the valves were organically diseased.

In all of these cases there was chronic disease of the valves. In five cases there appears to have been a fresh endocarditis engrafted upon the chronic affection, and it is possible that in some other cases this was present, but in the majority of the observations there was no acute endocarditis.

Adherent pericardium was noted in four cases, acute pericarditis in one.

It is evident that the association of thrombosis of the upper veins with valvular disease of the heart is almost, although not wholly, limited to cases of mitral disease, those of mitral stenosis, with or without insufficiency, taking the lead. The preponderance of females and of patients under middle age is thus, at least in great part, explained.

Relation to Rheumatism.—As is to be expected from its nature, the valvular lesion was most frequently caused by acute articular rheumatism, of which there was a distinct history in a little over half the cases, but other infectious diseases (scarlet-fever, pneumonia, influenza, puerperal infection, diphtheria, and chorea) also had a share in the causation. In a number of instances the valvular trouble seems to have developed insidiously. There is, therefore, nothing peculiar in the antecedent histories.

Although several observers regarded the occurrence of the thrombosis as a direct manifestation of rheumatism, only in three cases was there any painful swelling of a joint present shortly before or during the attack of thrombosis.

Relation to Cardiac Insufficiency.—In the great majority of cases the thrombosis appeared during a condition of failure, generally extreme failure, of compensation of advanced mitral disease. The frequency with which pulmonary infarction was observed at autopsy is one of the many evidences of this. Relative insufficiency of the tricuspid valve and pulsation of the cervical veins were noted in many of the cases, and very likely were present in some of those in whose histories they are not noted, but it does not appear that these conditions, although contributory, are necessary factors in the causation of the thrombosis.

Location of the Thrombi.—A fact which at once arrests attention is that the thrombosis affected veins of the left side in 21 out of the 23 cases, of which 14 were unilateral and 7 bilateral. In only 2 cases were veins of the right side alone affected. Bilateral thrombosis is, therefore, more common than unilateral right-sided thrombosis, and unilateral left-sided thrombosis is by far the most common form of the disease. The hypotheses in explanation of this distribution will be considered later.

In 7 cases the thrombosis was apparently limited to the arm veins, in 1 to veins of the neck, and in 15 cases veins both of the neck and arms were thrombosed. In 1 case the thrombus was confined to the left subclavian vein, in 1 to the left internal jugular, and in 2 to the left brachial. In all the other cases more than one vein was thrombosed, the combination varying in different cases, the most common being continuous thrombosis of the left innominate, internal and external jugular, subclavian, and axillary veins. The superior vena cava was partly or wholly thrombosed in 6 cases (confirmed by autopsy in 4), the innominate in 11 (confirmed by autopsy in 8), the internal jugular in 13 (autopsy in 10), the external jugular in 12 (autopsy in 8), the subclavian in 15 (autopsy in 12), the axillary in 10, the brachial in 5, the inferior thyroid in 1, and the left lateral sinus of the brain in 1.

The commonest starting-point for the thrombus was the lowest part of the left internal jugular (bulb), or of the left external jugular vein and the left innominate or subclavian vein near the entrance of the jugulars. In many cases this was clearly demonstrated by the appearances of the thrombus and the venous wall in these situations. The thrombus may, however, originate in other veins, especially in valvular pockets of the veins in the left arm.

The most extensive thrombosis was that recorded by Poynton (Case 27), in which there was occlusion of the superior vena cava in its upper two-thirds, of both innominate, internal and external jugular, subclavian, and axillary veins, and the left inferior thyroid vein.

The thrombus was generally a continuous one, apparently originating in one vein, whence it was propagated into peripheral veins and also centrally, even into the superior vena cava.

Characters of the Thrombi and Venous Walls.—The thrombi were mixed, the prevailing color being dark red. The older parts were gray or reddish-gray and adherent. The fullest description of the microscopical characters is that which I have recorded under Case 1. In a few instances the thrombus was centrally softened, but in most it was solid and completely occluding except at the ends and except in the mural thrombus mentioned in Case 27. The older thrombi were usually more or less advanced in organization. Bacteria were searched for in apparently only 3 cases, and were found only in my Case 1, the organism here being Streptococcus pyogenes.

In 6 cases it was noted that the wall of the thrombosed vein was thickened, but this thickening was generally regarded as secondary to the formation of the thrombus, which was two or more weeks old. Only in Helen Baldwin's case is it distinctly stated that the appearances indicated phlebo-sclerosis (subclavian vein) antedating the thrombus. In two of Poynton's cases the jugular and innominate veins, where the oldest part of the thrombus was situated, were narrowed, white, and firm, and a similar condition was noted by Robert. Chronic disease of the veins evidently plays only a very minor part in the causation of the thrombosis in these cases.

Effects and Symptoms.—The anatomical lesions outside of the vessels directly referable to the thrombosis were œdema of the tissues and swelling of the lymphatic glands. The œdema cannot always be explained entirely as the result of venous congestion, but is in part inflammatory, as is true in general of the œdema in phlegmasia. Swollen lymphatic glands, which were sometimes hæmorrhagic, were often observed in the neighborhood of the thrombosed veins. This swelling was generally acute and evidently secondary to the thrombosis,

but Kahn attributed the exceptional localization of the thrombus in the right subclavian vein in his case (No. 19) to the pressure of previously enlarged glands. Poynton noted cerebral œdema in one of his cases which had manifested brain symptoms.

The local symptoms are the usual ones of venous thrombosis: pain, tenderness, œdema, the presence of hard, sensitive venous cords, and distension of superficial veins. Often the pain, sometimes the œdema, was the first symptom. The œdematous swelling may be hard and brawny, or softer as in dropsy. In some cases the pain and œdematous swelling were first manifest in the neck, in others in the arm. Extension of the œdema to the front and side of the upper part of the thorax was repeatedly noted. Occasionally the side of the face corresponding to the thrombosis was œdematous. The extent and distribution of the œdema, while dependent in the first instance upon the extent and location of the thrombosis, were influenced, as is usual in venous thrombosis, by other factors, so that they varied much in cases with the same distribution of the thrombi.

Constitutional symptoms of the thrombosis, if present, could not readily be separated from those of associated conditions. The marked difference between the buccal and rectal temperatures observed in Helen Baldwin's case (No. 21) is interesting. Cerebral symptoms, attributed by Poynton to cerebral œdema, were observed in a few cases.

Hanot in 1874 called attention in a short note to the more rapid onset and the longer persistence of œdema of the left arm, as compared with the right, in cardiac affections, and he explained this peculiarity by the greater length and obliquity of the left innominate vein. It would appear, therefore, that œdema limited to or in excess in the left arm in heart disease should not be regarded as positive evidence of thrombosis of veins conveying blood from this extremity. While this is doubtless true, it is desirable, in view of the cases reported in this paper, to search carefully in these instances for other evidences of thrombosis.

Prognosis. — Of the twenty-three cases, nineteen ended fatally and four (Cases 2, 6, 12, and 20) recovered. The gravity of the prognosis doubtless mainly results from the circumstance that the occurrence of the thrombosis is in itself an index of extreme failure of compensation of the valvular lesion, being sometimes scarcely more than a terminal event. We know that occlusion of the superior vena cava and its large tributaries, due to other causes, may be completely compensated by the development of a collateral circulation, and there are instances of this even in the present group of cases (Nos. 6 and 20), so that it is less the venous occlusion than the condition of the heart and the frequent

presence of pneumonia or other terminal infections which makes the issue so unfavorable. In rare instances, thrombosis of the upper veins may occur at a period when the compensation is not badly broken (Case 2). Large pulmonary embolism was not observed. Whether pulmonary infarcts, which were frequently present, were attributable to emboli derived from the venous thrombi, was not determined. The duration in the fatal cases of thrombosis varied from two days to six weeks.

Causation. — Although five of the cases were reported as instances of rheumatic phlebitis, it does not seem to me demonstrated that even in these cases (Nos. 11, 18, 25, 26, 27) this was the correct explanation, and it is certain that the great majority of cases cannot be explained in this way. It is true that phlebitis, or venous thrombosis, is a genuine, although infrequent, complication of acute rheumatism, and there is some evidence that it may affect veins of the upper extremities somewhat more frequently than does thrombosis due to most other causes, but there is no such enormous preponderance of upper thromboses as in the class of cases now under consideration. As already mentioned, the histories of the cases do not support the view that rheumatism had more than a minor share in the immediate causation of the thrombosis. There is no reason, however, why acute rheumatism, like other infections, may not directly participate in the causation of the venous thrombosis.

French writers (Peter, Parmentier, Kahn, Huchard) attribute the thrombosis to cardiac cachexia combined with circulatory disturbances. They say that this cardiac cachexia (so designated by Andral) is something to be distinguished from asystoly, that is, from mere breakage of compensation. They bring this class of thromboses, therefore, into line with that complicating tuberculosis and cancer, and explain the peculiar localization by the particular disturbances of the circulation in cardiac disease. The underlying cause, according to this view, is some alteration in the chemical composition of the blood. It is difficult to say how much weight is to be attached to this explanation. In many cachectic conditions there is an increase in the blood platelets, attributable probably to weakened resistance of the red corpuscles, and some writers have brought cachectic thromboses into relationship with this increase. According to van Emden, the number of platelets is diminished in the chronic congestion of heart disease. I know of no observations concerning the number of platelets in these cases of heart disease with venous thrombosis. I could find nothing in the histories of most of the cases indicating any peculiar cachexia, and I am not inclined to regard this explanation of the thrombosis of heart disease as a satisfactory one.

The first thing which needs explanation is the localization of the thrombosis in the veins receiving blood from the upper part of the body, and especially from the left side. Hanot and Parmentier explain the preference for the left side by the greater length and obliquity of the left innominate vein, so that, like the return flow of blood from the left leg, that from the left arm and left side of the neck is more difficult than from the right side, this difficulty being, of course, most in evidence in the venous congestion of uncompensated cardiac disease. Hirschlaff suggests that an additional factor may be the greater frequency of imperfect development and of insufficiency of the valve near the junction of the internal jugular and subclavian veins on the left than on the right side. To these factors I would add pressure, either direct or indirect, on the left subclavian vein from the dilated left auricle and dilated large pulmonary vessels. Popoff noted pressure from this source on the left subclavian artery in mitral stenosis with insufficiency as a cause of relative weakness of the left radial pulse (pulsus differens). I would refer to his article for a consideration of the factors concerned in producing this pressure.

Peter, in 1873, was the first to suggest that the frequency with which the thrombus starts from the lower ends of the jugulars is to be explained by the presence of valves in this situation. I am inclined to lay emphasis upon the readiness with which an eddying or whirling motion of the blood may be set up in heart disease in the lower ends of the jugulars and the adjacent parts of the innominate and subclavian veins. Von Recklinghausen has brought forward strong evidence that this whirling movement (Wirbelbewegung) of the blood is of great importance in determining the localization of thrombi. The valves, the bulbous enlargement at the lower end of the internal jugular, the attachment of the veins to fasciæ, and the coming together here at oblique and right angles of currents of blood with different pressures and velocities, seem well calculated to cause in these veins whirling or vortical motion of the blood current, especially in the circulatory conditions of broken compensation of mitral lesions. Most favorable to this peculiar disturbance of the circulation would be tricuspid insufficiency with systolic reflux of blood into the veins. I would explain, therefore, the special localization of the venous thrombosis complicating cardiac disease, on the one hand by the particular disturbance of the circulation, and on the other by the anatomical disposition and structure of the veins.

These factors, however, explain only why certain veins are the seat of election for the thrombi. The circulatory conditions described must often exist within these veins in uncompensated cardiac diseases,

whereas venous thrombosis is a very rare complication of heart disease. In the case which first directed my attention especially to this subject, and which I have here reported as Case 1, an immediately exciting cause for the thrombosis was discovered by the demonstration of bacteria in the thrombus. This is the only case in the entire list in which micro-organisms were found, but so far as I can gather, in only three other cases were bacteria especially looked for, and it does not appear that in any of these latter were cultures made from the thrombus. Such cultures are, of course, necessary in order to exclude the presence of bacteria, and indeed our experience has been that even a negative result from cultures is not absolutely decisive. Evidence has accumulated in recent years in support of the infectious origin of many thrombi. Dr. Harris and Mr. Longcope in my laboratory have now examined bacteriologically forty-four thrombi, mostly peripheral venous thrombi of the so-called marantic type, and in thirty-four of these have demonstrated the presence of bacteria. As has been shown by Dr. Flexner in my laboratory, terminal infections are not uncommon in heart disease. The histories and autopsies of many of the twenty-three cases now under consideration revealed some infectious process, usually in the lungs, such as bronchitis, pneumonia, and pleurisy. While, therefore, it would be quite unwarrantable, from existing evidence, to refer this class of venous thromboses in cardiac disease positively to infection, this seems to me at present the most probable explanation.

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GENITO-URINARY TUBERCULOSIS: ITS DIAGNOSIS IN THE LABORATORY.

By FREDERIC E. SONDERN, M.D.

SINCE the discovery by Koch, eighteen years ago, that tuberculosis is invariably associated with the presence of a specific organism, the corroborative finding of tubercle bacilli in the secretions and excretions of the body has become a most important diagnostic factor. Tuberculosis in most organs is accompanied by a more or less conclusive clinical picture, but this I believe applies least of all to tuberculosis of the genito-urinary system. On this account it is extremely essential that laboratory methods, not only for the finding of tubercle bacilli in urine, but also for their differentiation from other bacilli, should be as perfect as possible.

For convenience, our subject may be subdivided as follows: First, the finding of tubercle bacilli in urine if present, and the prevention of erroneous conclusions as to the exact nature of the organism found. Second, corroborative evidences usually present in specimens of tuberculous urine, and the determination, as far as possible, of the probable seat of the tuberculous lesion.

The object of this communication is to call attention to a consideration of the above questions more from the laboratory standpoint than from the clinical one, but with the necessary clinical data, without which laboratory statistics are valueless.

I.—THE FINDING OF TUBERCLE BACILLI IF PRESENT, AND THE PREVENTION OF ERRONEOUS CONCLUSIONS AS TO THE EXACT NATURE OF THE ORGANISM FOUND.

Every general practitioner is able to demonstrate the presence of tubercle bacilli in tuberculous sputum, and as this procedure is so universally employed the technique has become a simple matter. For this purpose a considerable variety of methods have come into use, and all standard ones seem equally efficient in the task of demonstrating the bacilli. In urine, however, this is often a more complicated affair, as

the bacilli are present in much smaller numbers, and in demonstrating them, great care must be exercised in their differentiation from other bacilli which may at times resemble them. Many errors in diagnosis can be ascribed to the fact that the technique for staining tubercle bacilli in urine, and their differentiation, is not sufficiently detailed in many, if not most, of the text-books on laboratory methods. The only organisms which may be mistaken for tubercle bacilli ordinarily met with in the microscopic examination of urinary sediments are smegma bacilli, and a more detailed consideration of this element of error is of great importance, particularly as it is the point on which so little stress is laid in most of the text-books. Such books as those by Posner,[1] Sahli,[2] v. Kahlden,[3] Heitzmann,[4] Purdy,[5] Spaeth,[6] Rosenfeld,[7] and many others, while they detail more or less careful methods for finding tubercle bacilli in urine, do not mention the possibility of mistaking smegma bacilli for tubercle bacilli when searching for the latter. That the error is a common one can be concluded from the many cases reported in medical literature—for example, König, Bunge, Mendelsohn, and many others, where supposed tubercle bacilli found in urine led to a diagnosis not corroborated by subsequent developments, operation, or autopsy. In all the cases reported, both of urine and sputum, when the cause of error was made out, it was found that smegma bacilli had been mistaken for tubercle bacilli.

After having given careful attention to this particular line of work for quite a number of years, I certainly incline to the belief that the error in question must in the majority of instances be ascribed to inefficient methods of examination rather than to an actual inability to differentiate between the two varieties of organisms. That errors can occur even with the most painstaking care in examination cannot be denied, but usually this happens in exceptional cases only where a few isolated bacilli are found, and where for some reason the differential staining methods lead to no characteristic result. With improved technique it can be hoped that these elements of error will soon be eliminated. Schürmeyer's[8] statement that, when isolated bacilli only are present, no differentiation between tubercle and smegma can be made, is I believe too conservative.

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[1] Posner: *Diagnostik d. Harnkrankheiten*, 1896.
[2] Sahli: *Klinische Untersuchungsmethoden*, 1894.
[3] v. Kahlden: *Technik d. Untersuchung patholog. Präparate*, 1898.
[4] Heitzmann: *Urinary Analysis and Diagnosis*, 1899.
[5] Purdy: *Urinary Diagnosis*, 1898.
[6] Spaeth: *Die Chemische u. Mikroscopische Untersuchung d. Harns*, 1897.
[7] Rosenfeld: *Diagnostische Semiotik des Harns.*
[8] Schürmeyer: *Harnuntersuchung und ihre diagnostische Verwertung.*

The smegma bacilli are of no pathogenic importance, and deserve consideration only on account of being mistaken for tubercle bacilli. According to numerous published investigations, smegma bacilli are very commonly present about the genitals. Grünbaum [1] examined fifty specimens of urine from forty-seven individuals; the sediments obtained by centrifuge were spread on covers and stained with carbol-fuchsin. He found no smegma bacilli in the specimens from males, but found them in 59 % of the specimens from females. He found no smegma bacilli in urine obtained by catheter from eleven individuals including both sexes.

Bunge and Trautenroth [2] have not only found smegma bacilli about the genitals, but also on the surface of the body, in the nose and mouth, and in other cavities lined with mucous membrane; in the deep urethra in one out of twelve specimens, and almost constantly in the anterior urethra of healthy males; between the labia in twenty-eight out of thirty healthy women, usually in the vagina, and occasionally in the urethra. Specimens of urine drawn by catheter from the bladder were all negative.

Alvarez and Tavel [3] found smegma bacilli in thirty-three out of fifty primary syphilitic lesions, and in ten out of fourteen specimens of smegma obtained from healthy persons.

My personal experience with over six thousand specimens of urine stained with carbol-fuchsin allows the conclusion that more than 40 % of these presented smegma bacilli. Of this number comparatively few specimens were drawn by catheter, but in a very small number of these, few isolated organisms were found which were not tubercle bacilli but resembled them very closely, and were believed to be smegma bacilli on account of their staining qualities, although both Grünbaum [4] and Miller [5] deny their presence in bladder urine obtained by catheter. In 270 specimens of separately collected kidney urine, all of which were obtained by the ureter-catheter excepting a few by the Harris segregator, organisms believed to be smegma bacilli were observed in a few instances. These observations have led me to abandon the conclusion advocated by Miller [6] and others, that urine obtained by catheter from the bladder contains no smegma bacilli, and justify the opinion that these specimens should have the same care in differential staining as those voluntarily voided.

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[1] Grünbaum: *Lancet*, 1897.

[2] Bunge and Trautenroth: *Fortschr. d. Med.*, 1896.

[3] Alvarez and Tavel: *Arch. d. Phys.*, 1885.

[4] Grünbaum: *l. c.*

[5] Miller: *Medicine*, Detroit, 1898.

[6] Miller: *l. c.*

Smegma bacilli present wide variations in size and form to which Hueppe [1] and others call attention; so much so, that I believe it is but reasonable to question if all these are really one species of organism. True, they all show the same staining qualities, but other organisms rich in fatty matter may do this; and they do vary greatly in the time required to decolorize them by the various methods. Thus it is that only certain varieties of smegma bacilli resemble tubercle bacilli in outline. While it is true that successful culture attempts have been reported, these, however, lack anything like universal corroboration and suggest the idea that at the present time the morphological status of the organism is not yet established.

Smegma bacilli may occur in clumps or singly. When in clumps the arrangement is usually an irregular one, for example such as seen in a Widal clump reaction in typhoid cultures; but I have never found them in the characteristic grouping to which I will refer later when considering tubercle bacilli. Not only should the foregoing be considered an important differential point, but also the fact that with proper technique, tubercle bacilli usually present a beaded appearance or vacuoles, a constant feature in at least some of the tubercle bacilli, and *never present* in smegma bacilli. The former feature, finding bacilli in clumps, is referred to by Bunge and Trautenroth [2]; they state that smegma bacilli are often found in clumps, thus resembling tubercle bacilli; as explained above, when they are found thus, the grouping is always an irregular one, never presenting the characteristic picture almost always noted when dealing with tubercle bacilli. Grünbaum [3] also states that smegma bacilli show a grouping not seen with tubercle bacilli, while my experience, as stated, is just the opposite, *i. e.*, tubercle bacilli usually show characteristic grouping, while smegma bacilli, if in clumps, show no typical arrangement.

Tubercle bacilli found in urinary sediments, when properly stained, occur singly at times, but in the large majority of cases in smaller or larger groups. The grouping is generally characteristic—the bacilli lie side by side parallel, and end to end in a more or less regular arrangement; the whole group is usually curved, as for example the whole or a part of the letter "S." This typical arrangement is described by v. Jaksch [4] and is pictured in some of the other text-books, although not mentioned in the description.

Differential Staining: It is universally conceded that all the stand-

[1] Hueppe: *Methoden d. Bakterienforschung* 1891.

[2] Bunge and Trautenroth: *l. c.*

[3] Grünbaum: *l. c.*

[4] v. Jaksch: *Klinische Diagnostik*, 3 Auflage.

ard methods for the differential staining of tubercle bacilli will also stain smegma bacilli; so that these usual methods are of no avail in distinguishing between at least these two varieties of organisms. The opinions of Lenhartz,[1] Pappenheim,[2] and others, that the Gabbet method is quite unsuited as a differential stain for tubercle bacilli in urine, are certainly correct, although the method is recommended by many.

Bunge and Trautenroth[3] recommend that specimens be spread on covers and fixed by heat, then deprived of fatty matter by immersion in absolute alcohol for three hours; this to be followed by a 5% solution of chromic acid for fifteen minutes, then washed in water, stained with carbol-fuchsin, decolorized with a dilute sulphuric acid solution for three minutes, pure nitric acid for one to two minutes and further decolorized and counterstained with a concentrated alcoholic solution of methylene blue for five minutes. Pepper and Stengel[4] and Miller[5] doubt the efficacy of this method, which views I share after repeated trials with it. A. Pappenheim[6] recommends that specimens should be stained with hot carbol-fuchsin in the usual way; the excess of stain is then drained off, and the specimen decolorized and counterstained by immediately dipping it three to five times into the following solution: In 100 parts of absolute alcohol dissolve one part coralline; this solution is saturated with methylene blue (requires a large amount); then twenty parts of glycerine are added. The specimen is then washed in water and dried. Duration of manipulation three minutes. Tubercle bacilli are stained red, smegma bacilli blue. My first few attempts with this method were so discouraging that I abandoned it, but I would distinctly state that the trials were not sufficient to allow any opinion on its merit. Nor have I had any degree of success with the Grethe method as advised by Simon.[7] Specimens are stained with alcoholic fuchsin, washed in water, decolorized, and counterstained with a concentrated alcoholic solution of methylene blue.

Staining with carbol-fuchsin, decolorization with nitric acid and subsequently with absolute alcohol, is the method which I have found most useful, though by no means free from objections. The specimens should be dried preferably on slides instead of cover glasses; and fixed

[1] Lenhartz: *Münchener med. Woch.*, 1897.
[2] Pappenheim: *Berliner kl. Woch.*, 1898.
[3] Bunge and Trautenroth: *l. c.*
[4] Pepper and Stengel: *Amer. Year Book of Med. and Surg.*, 1898.
[5] Miller: *l. c.*
[6] Pappenheim: *l. c.*
[7] Simon: *Clinical Diagnosis*, Phila., 1897.

with heat, but *overheating must be carefully avoided because this no doubt increases the power of resistance of smegma bacilli toward the decolorizing agents.* The slides are then stained in the usual way with carbol-fuchsin and heat for five minutes; here again overheating must be avoided for the same reason; the stain should steam slightly, but not boil. A small water-bath constructed for the purpose has been found very useful in my laboratory. The excess stain is then washed off with water and the specimen well decolorized with one to six solution of nitric acid. On microscopic examination everything except the bacilli should have lost color completely. A few of the best groups of bacilli are selected and "ringed" by a substage or nose-piece arrangement, of which there are a number in the market; so that the same groups of bacilli can easily be found again. Then the specimen is immersed in absolute alcohol for further decolorization. While it is a well-established fact that smegma bacilli are decolorized by absolute alcohol much more quickly than tubercle bacilli, the objectionable feature of the method is, that no definite time for this immersion can be stated in which the smegma bacilli will be completely decolorized, with at the same time no action on the color of tubercle bacilli. While Miller[1] states that the specimen should remain in the absolute alcohol for at least five minutes, I have not found this nearly long enough. Ewing[2] advises the use of 95% alcohol for eight to twelve hours, which is much more satisfactory. After considerable experimental work, I find that in the large majority of specimens, provided the sediment was obtained from an acid urine, an immersion in absolute alcohol from five to eight hours will completely decolorize the smegma bacilli without materially affecting the color of tubercle bacilli; in fact the latter have frequently withstood the action of absolute alcohol for twelve hours. Smegma bacilli decolorize much more quickly in some specimens than in others. Tubercle bacilli in different specimens also vary as to their ability to withstand the decolorizing effect of absolute alcohol. Why this should be so seems difficult to explain, except that the reaction of the specimen of urine from which the sediment was obtained does exert an influence. Stained tubercle bacilli in alkaline specimens, and particularly if an ammoniacal fermentation is present, possess less resistance to alcohol than those of acid specimens. Bunge[3] has also called attention to this point in his article.

Animal Inoculation: Sediments of urine containing tubercle bacilli, when inoculated into guinea-pigs, lead to the development of

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[1] Miller: *l. c.*

[2] Ewing: *Clinical Diagnosis*, 1898.

[3] Bunge: *l. c.*

tuberculosis and death. The autopsy usually presents typical pictures which establish the diagnosis beyond a doubt. This method *if combined with the proper precautions* is certainly a valuable one. It at times happens, however, that the animals die as the result of other toxic or bacteric infections, and if enlarged mesenteric glands only are found, a tuberculous infection must not be assumed until a microscopic examination of these glands reveals a tuberculous lesion. On several occasions I have had inoculations of doubtful tuberculous urine made and the animals died without, however, presenting typical post-mortem pictures of tuberculosis. Early in November, 1899, I received a specimen of urine for analysis, with a clinical history as follows: Young unmarried woman, nocturnal frequency of micturition for some months, no tenesmus. General health good. No tubercular family history. The urine on examination presented evidences briefly as follows: Small daily amount, faintly acid reaction, high gravity, normal daily excretion of solids, evidences of some hyperæmia of the renal parenchyma, nothing pointing to a lesion of the renal pelvis, and elements of some chronic cystitis, no blood. Staining revealed few isolated bacilli. Various methods to differentiate between smegma and tubercle bacilli were employed, but the outcome was at best a doubtful one, still I strongly suspected that they might be tubercle bacilli. A fresh specimen was voided into a sterile vial, immediately sedimented by centrifuge in sterile tubes, and inoculated, by Dr. A. V. Moschcowitz at the Pathological Laboratory of the College of Physicians and Surgeons, New York, into two guinea-pigs, one subcutaneously, the other intraperitoneal. The former animal died on the thirteenth, the latter on the eighteenth day. Dr. Moschcowitz also had the kindness to make the post-mortem examinations, which revealed pictures alike in both animals: marked enlargement of the mesenteric glands and other lymph nodes usually considered typical of an early tuberculosis. On microscopic examination, a hyperplasia of the lymph node was found, but absolutely no evidences of a tuberculous lesion. I would add that the patient recovered from the cystitis and the secondary renal hyperæmia on appropriate treatment. This case teaches conclusively the care necessary to make an inoculation test trustworthy.

Culture Methods: The usual culture methods employed for the growth of tubercle bacilli in scientific research are not applicable for purposes of diagnosis of these bacilli in urine, because their growth is very slow and is buried under the relatively rapid growth of contaminating organisms present in every specimen of urine. Purdy[1] advises cultures

[1] Purdy: *l. c.*

in gelatin and subsequent inoculation, but does not detail his method of doing away with the contaminating organisms.

W. Hesse,[1] in the latter part of 1899, announced a new method for the rapid growth of tubercle bacilli, which promises to be an important aid in diagnosis, and I believe in their differentiation from smegma bacilli as well. The culture medium is made up as follows:

Nährstoff Heyden	5.0
Sodium chloride	5.0
Glycerine	30.0
Agar-agar	10.0
Normal solution Soda crystals...28.6 : 100	5.0
Water to	1000.0

This medium is used for plate cultures which after inoculation are kept in the incubator at a temperature of 37° C. According to Hesse's article, he worked chiefly with sputum and found evidences of growth of the tubercle bacilli in five to six hours, and decided colonies in from twenty-four to forty-eight hours. The contaminating organisms are retarded in their growth. Wilson[2] and Hiss[3] have endorsed Hesse's claims to a certain extent, but do not mention any trials with urine containing tubercle bacilli. Since the early part of December, 1899, I have applied the method to almost every specimen of urine sent me for examination which might possibly contain tubercle bacilli, or which presented tubercle bacilli or smegma bacilli on microscopic examination, and for the present have arrived at the following conclusions: As yet the culture method has never revealed tubercle bacilli in specimens in which the same were not found by some other method of examination, excluding animal inoculation. In a moderate number of the specimens the rapid growth of contaminating organisms ruined the culture attempt. Numerous specimens were encountered, however, in which the usual microscopic examination revealed so very few bacilli not always characteristic, while the Hesse culture method produced pictures justifying an undoubted diagnosis of tuberculosis. All attempts, and they were numerous, to grow smegma bacilli from urine and from smegma obtained from both male and female resulted negatively. If the above conclusions are found to hold good after more extensive trial, the method will certainly be of value in the differentiation between tubercle and smegma bacilli, and will furnish a more suitable product for animal inoculation.

Additional: Urinary sediments intended for staining for tubercle

[1] W. Hesse: *Zeitsch. f. Hygiene u. Infectionsk.*, vol. xx., p. 151, 1899.

[2] Wilson: Report N. Y. Path. Soc., *Med. Record*, March 3, 1900.

[3] Hiss: Report N. Y. Path. Soc., *Med. Record*, March 3, 1900.

bacilli should preferably be obtained by centrifuge, because this not only permits examining the specimen as fresh as possible, but also usually results in the presence of larger numbers of organisms.

When the sediment of pus is very thick and profuse, and the bacilli found in very small numbers, it is well to employ the Biedert or the v. Sehlen method, which will result in the presence of larger numbers of bacilli, but makes differentiation of tubercle and smegna bacilli more difficult, as it disturbs the characteristic grouping and affects to a certain extent the ability on the part of tubercle bacilli to retain the stain while immersed in the absolute alcohol.

II.—CORROBORATIVE EVIDENCES USUALLY PRESENT IN SPECIMENS OF TUBERCULOUS URINE, AND THE DETERMINATION AS FAR AS POSSIBLE OF THE PROBABLE SEAT OF THE TUBERCULOUS LESION.

Volume Voided in Twenty-four Hours: The average volume of urine in twenty-four hours in seventy-four cases of undoubted genito-urinary tuberculosis was 1430 c.c., the largest amount 2200 c.c., the smallest 720 c.c.; the former was a tuberculous pyelo-nephritis, the latter a tuberculous lesion of the prostate and bladder. In general it may be stated that in tuberculous lesions of the kidney and its pelvis, the daily excretion is increased as in like lesions due to other causes. The frequency of micturition without tenesmus seems to be out of proportion to the polyuria, and chiefly nocturnal.

Reaction: While Purdy[1] claims that these specimens are usually alkaline, most other authors remark that an acid reaction is the rule: over 97% of my specimens were decidedly acid.

Rovsing calls attention to the fact that when a specimen contains evidences of a marked chronic cystitis and presents an acid reaction, tubercle bacilli should be searched for. I have frequently justly suspected specimens of this kind, where the clinical history did not suggest a tuberculous process.

Amount of Albumin, Urea, Chlorides, etc.: These correspond to the lesion in question and present nothing characteristic.

Microscopic Examination: Blood: At least a few blood corpuscles are almost always present. Blood cells were found in 98% of my specimens. *Pus:* At least some pus cells and usually smaller or larger amounts of pus were found in all the specimens even when the tuberculous lesion was but a comparatively slight one. The only specimens which contained tubercle bacilli and no pus also presented no evidences

[1] Purdy: *l. c.*

of a genito-urinary lesion, and the bacilli were referable to a miliary tuberculosis elsewhere, or to a pulmonary tuberculous process, the bacilli having, so to speak, filtered through the kidney.

Casts : Usually the severity of the lesion of the renal parenchyma is indicated to a certain extent by the casts present; in these cases, however, this is no characteristic feature, being the same as would be observed in like lesions due to other causative factors. Frequently, however, rather extensive tuberculous lesions of the kidney may be present and at the same time relatively few or no casts found, which may possibly be explained by less hyperæmia of the parenchyma in tuberculous lesions than is usually found in the similar lesions due to other causes. Especially where the kidney and its pelvis present many small tuberculous nodules and there are perhaps only several small abscesses discharging into the urinary tract, casts are oftentimes absent from the urine, or there may be a very few hyaline casts only present.

Bacteria : The presence of tubercle bacilli and their demonstration in these specimens we have considered. While these specimens always show the non-pathogenic organisms usually found in urine, it is a noteworthy fact that in the large majority of instances they do not seem subject to the early development of either an acid or an alkaline fermentation. I have very frequently observed that perfectly normal specimens of urine exposed to room temperature usually undergo decomposition of one or other kind much more quickly than a tuberculous urine, while we would certainly expect the opposite to be the case, owing to the presence of so much more organic matter. What the agent may be that is the cause of this preservation I do not know, but believe it reasonable to suppose that it is due to some toxin.

Epithelium : The epithelial cells present may under circumstances be taken as a partial indicator of the seat of the lesion, as far as this is permissible in like lesions due to other causes.

Crystalline and Amorphous Deposits : It very frequently happens that with the acid reaction usually present there are few uric-acid crystals in the sediment or small amounts of urate salts, but a deposit sufficiently marked to create a suspicion of stone I have very rarely found. Oxalate of lime deposits also seem very infrequent and were not found in any of my specimens. Triple phosphates and other deposits usually present in ammoniacal urine will of course be found when these specimens have undergone that decomposition.

Résumé.

Urinary Sediment to be Stained for Tubercle Bacilli :

Specimen as fresh as possible.

Sediment obtained by centrifuge; if thick and purulent, prior use of Biedert or v. Sehlen method when necessary.

If not sufficient albumin, add small amount of dissolved egg albumin.

Sediment dried on microscopic slides at room temperature, under cover.

Fixed by heat, carefully avoiding overheating for reason stated.

Stained with carbol-fuchsin and heat, again avoiding overheating.

Washed in water and thoroughly decolorized with nitric acid 1 : 6.

Examined with microscope and several good groups of bacilli "ringed."

Immersed in absolute alcohol for five to eight hours, which almost always decolorizes smegma bacilli and does not affect color of tubercle bacilli.

If in doubt, use Hesse culture method.

If desired, the colonies thus obtained can be inoculated into guinea-pigs and enlarged lymph nodes present on post-mortem examination looked over for characteristic tuberculous lesions.

Miliary Tuberculosis Outside of the Genito-Urinary System and especially Tuberculous Pulmonary Lesions: The urine in these cases sometimes contains a few isolated tubercle bacilli, which seem to have filtered through the kidneys, so to speak, occasionally accompanied with evidences of a very slight renal hyperæmia; *i. e.*, no polyuria, oftentimes the specimen is somewhat concentrated, normal daily excretion of solids generally, slight traces of albumin, very few hyaline and occasionally an epithelial cast, no pus or blood. In cases of this kind with a urinary condition as above, one is oftentimes prone to assume extension of the tuberculous process to the genito-urinary system before it has really occurred. This is especially the case, as it is difficult to draw a sharp dividing line between specimens of these cases and those obtained from the following group of cases to be considered.

Renal Miliary Tuberculosis or Tuberculous Lesions of the Kidney without Degenerative Changes in the Deposits: These specimens frequently show the same urinary picture as detailed above, with the exception that more or less polyuria is always noted, the clinical symptom — nocturnal frequency of micturition — is almost constant, and the presence of at least few blood and pus cells, and epithelium presumably from the renal pelvis, the rule. These blood and pus cells are not numerous enough to infer a pyelitis, their presence being, I believe, referable to a hyperæmia of the lining membrane of the renal pelvis. The cases met with of course occur in all grades, from the slight ones just described to those in which a large part of the parenchyma is

destroyed, and where the urinary picture becomes that seen in chronic nephritis of the contracting kidney type — polyuria, diminution in the daily excretion of solids, particularly urea, more or less albumin, a smaller or larger number of casts, with the same few blood and pus cells and epithelium referable to the renal pelvis.

Renal Tuberculosis with Degenerative Change in the Tuberculous Deposits, these Abscesses Communicating with the Renal Pelvis: These are the specimens of urine which are most frequently received for analysis. They present the typical picture of a tuberculous pyelo-nephritis, and are the ones in which tubercle bacilli are most readily found. Owing to the associated and more or less pronounced cystitis, with at times alkaline fermentation, these specimens resemble those from like lesions due to other causes, and mistaking smegma bacilli for tubercle bacilli in the latter is the most frequent cause of an erroneous diagnosis of tuberculous pyelo-nephritis.

A typical specimen from a case of tuberculous pyelo-nephritis of this class presents the following picture: more or less polyuria, lowered specific gravity, an acid reaction, rather pale color, a not offensive odor, an amount of albumin corresponding to the degree of renal involvement in addition to what the pus, etc., would account for, possibly traces of acetone, daily amount of solids, particularly urea, depending on the degree to which the parenchyma is involved, and a more or less profuse sediment, showing no tendency to coagulation; on microscopic examination, almost invariably a small amount of blood, more or less pus according to the degree of pyelitis and the number of tuberculous abscesses, usually few casts only, irrespective of the extent to which the parenchyma is involved, and epithelial cells referable to the renal pelvis. In addition there are elements of more or less cystitis, but usually these are insignificant as compared to the main lesion. It is only in exceptional and usually advanced cases that the evidences of chronic cystitis are decided, and an ammoniacal fermentation results. Tubercle bacilli are usually easily found, and if there is no decomposition, they are without difficulty differentiated from smegma bacilli by appropriate methods. If the cystitis is not decided, it is strange how long the specimen may stand at room temperature, before decomposition occurs.

A vital question remains—to what extent does the usual analysis justify an opinion as to the involvement of one or both kidneys? If the specimen presents evidences of decided tuberculous degeneration of the kidney and the daily excretion of solids, particularly urea, remains perfectly normal, it may be reasonable to infer that *possibly* the other kidney is not affected and is doing the excretory work for both.

A more definite conclusion is, I believe, not prudent, and absolute information on this point must be left to the cystoscopic examination and the analysis of separately collected urines obtained by ureter-catheter, for those collected with the Harris segregator seem unreliable for this purpose, as the horns of the instrument may detach tuberculous masses from the walls of the bladder, which might be looked upon as being of renal origin.

Vesical Tuberculosis alone, or Associated with a Tuberculous Lesion of the Prostate, Seminal Vesicles, etc.: To judge by the specimens received, the former condition is very rare, the latter more frequent, but usually they seem to be the result of a primary renal lesion. When existing alone, the only characteristic features found in the urine are tubercle bacilli, with usually an acid reaction, the specimens otherwise resembling those found in lesions of the same organs due to other causes.